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THE THORACIC INDEX

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1. STATEMENT OF PROBLEM

THE thoracic index of man is the breadth of the chest at the level of the nipples, divided by the antero-posterior diameter (chest depth) at the same level. Calling chest depth 100 the transverse diameter in man is of the order of 135. This quotient is the *thoracic index*.

Our problem is: How does the thoracic index change with age, sex and race; and what interpretation is to be given to the marked ontogenetic changes?

2. METHODS AND MATERIAL

In making the measurements on the living to determine thoracic index certain technical matters must be considered. The thoracic breadth (= transversal Brustdurchmesser; frontale Brustweite; largeur de la poitrine) is intended to be Martin's ('28, p. 159) measurement No. 36. This is defined as: "Direct measurement. Straight line distance from one another of the two most laterally expanded points on the ribs at the level of the mesosternale." Duckworth's ('19, p. 65) translation of the International agreement made at Monaco states: "27. antero-posterior diameter of the thorax (No. 1). This measurement is to be made in the same horizontal plane as is defined in the case of the transverse diameter of the thorax No. 1 ['in the horizontal plane at the level of the base of the ensiform cartilage']." I have been in the habit of holding the anthropometer rod at the level of the nipples in front, letting

the arms of the rod fall across the widest part of the thorax in the vicinity of the 6th or 7th rib; whichever is a maximum (Figure 1).

The chest depth, or antero-posterior diameter, is approximately Martin's No. 37 "(Sagittaler Brustdurchmesser; gerader Brustdurchmesser, Brusttiefe, sagittale Brustweite, épaisseur du thorax). Direct measurement." This is further defined as: "Direct distance of mesosternale, or better the lower margin of the corpus sterni, from the vertebral spine of the dorsal vertebra which lies in the same horizontal plane, in quiet breathing. Arms hanging quietly by side. Large 'Tasterzirkel.' Care should be taken that the instrument is held strictly horizontal." It must be said that my technique is in some respects nearer Martin's 37a or more strictly Sargent's technique; namely: The one arm of the calipers at a point of the sternum at the level of the nipples; and the sagittal diameter measured *perpendicularly to the long axis* of the vertebral column at this level (Fig. 1). In making this measurement I formerly used the straight-armed calipers, or "Stangenzirkel." For young children these serve to measure the perpendicular distance from sternum to dorsal vertebra. But as my children have grown up, development of pectoral muscles and breasts has made necessary the use of the large "Tasterzirkel" or upper section of anthropometer with curved arms. As my earlier technique (that upon which the present data are based) tended to prolong the anterior extremity of the dimension and to shorten the posterior extremity (as compared with No. 37) the deviation from the value of No. 37 is very small. I feel strongly that the Sargent dimension, with the use of the "Tasterzirkel," is to be preferred to a strictly horizontal measurement. For, depending on the degree of inclination of the dorsal vertebral series, the horizontal dimension is more variable than the perpendicular one. Papillault ('06, p. 63) has made this point.

The children measured were the same as described in earlier papers (Davenport, '32, '33) from the Orphan Asylum of Brooklyn (BOA) and from Letchworth Village (LVD). The measuring was done by myself, with the aid for the most part, of Mrs. T. Laanes. The data were worked on and the indices computed by Mr. W. Drager and Miss C. Carley. The children were measured repeatedly once or twice a year and sometimes more frequently.

The definition of the thoracic index given in the first paragraph is that specified by Martin ('28, p. 174). He adds: In relation to human

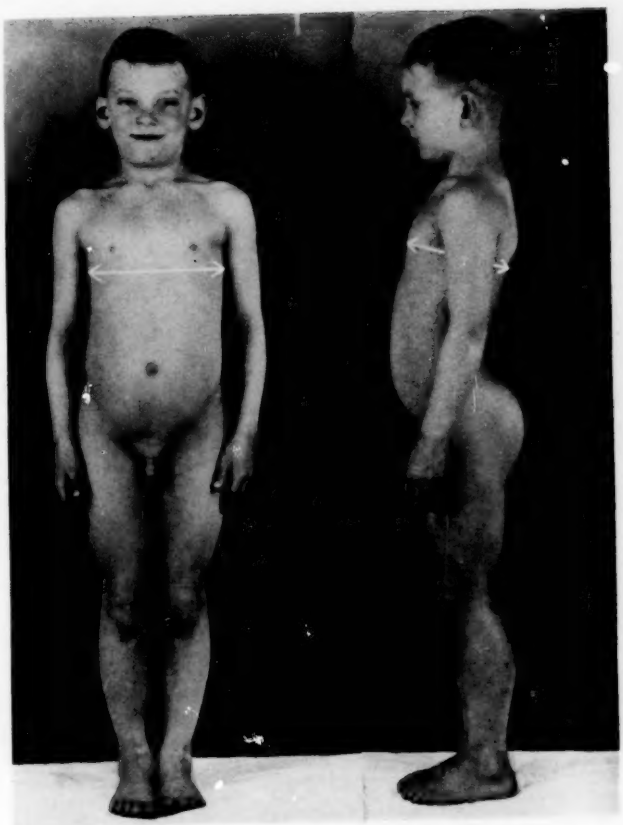

FIG. 1. PHOTOGRAPH OF A BOY (W. L. LVD No. 68) SHOWING POINTS BETWEEN WHICH TRANSVERSE CHEST DIAMETER AND ANTERO-POSTERIOR CHEST DIAMETER WERE TAKEN

conditions the index is computed in inverted fashion, though, with comparative studies in view, the computation as given is to be preferred.

Unfortunately, the comparison of the "thoracic index" of different authors has been rendered difficult by different methods of computing it. The history of the thoracic index may be worth tracing. Fourmentin (1874, p. 19) seems to have been the first to use the term. He says:

> J'ai appelé . . . . indice thoracique centésimal le rapport du diamètre maximum transverse au diamètre antéro-postérieure maximum. J'ai pris les dimensions correspondantes à celles de l'indice céphalique, afin qu'on put, au besoin, comparer les deux indices; il en est résulté que, la dimension transversale étant ici la plus grande, l'indice thoracique obtenu est plus grand que 100.

It is thus quite clear that Fourmentin knew exactly what he was doing and had a reason for computing the index in that way.

Weisgerber (1879) undertook a comparative study of the thoracic index in mammals, apparently at the instigation of Broca, in 1877. He computed the thoracic index precisely as Fourmentin had done and comments on it in the same way. He worked much on the skeletons of various mammals. He measured the external dimension of the thorax by means of a graduated large "compas d'épaisseur of Boudeloque" (essentially a pelvimeter) reaching from the lower part of the sternum to the summit of the spine of the vertebra nearest to the horizontal passing through the first point. The transverse diameter passed through the seventh rib so as to give a maximum.

Weisgerber measured also the internal dimension of the thorax (from the articulated skeleton). For this he used a "compas ordinaire," which I interpret to mean a large straight-legged compass of the type used by draftsmen. Just how the internal dimension is taken I cannot find that he states; probably on the inside of the skeleton axad of the points to which the external dimensions are measured.

So far everything is harmonious. But in 1886, Hovelacque et Hervé in their *Précis d'Anthropologie, Paris,* 1886, took the antero-posterior diameter as numerator. Also, Rodes ('05), Papillault ('06), Jackson ('07), and others have used the reciprocal of Fourmentin's index. Other authors have used, some the standard and some its reciprocal, so that much confusion and the necessity for transforming one index into the other has resulted. It is hoped that there will be a return to the standard set by Fourmentin and adopted by Martin.

Age Changes in the Thoracic Index

Data concerning prenatal changes in the thoracic index have been published by a number of authors. Some of them are given in Table 1, all indices being reduced to the formula: breadth ÷ depth.

The thoracic index was found by Godin for his French boys of 13.5 to 17.5 years, measured repeatedly. He found indices as follows:

AGE	13½	14	14½	15	15½	16	16½	17	17½
Thoracic index ...	137	139	135	137	135	137	133	135	135

The series shows a decrease from 14 years onward, that is, the chest is getting rounder in the older boys.

The change in form during development, especially the increase of index from 55 to 135, is a striking fact that has been discussed by Wiedersheim (1928) and Schultz (1924). (Fig. 2)

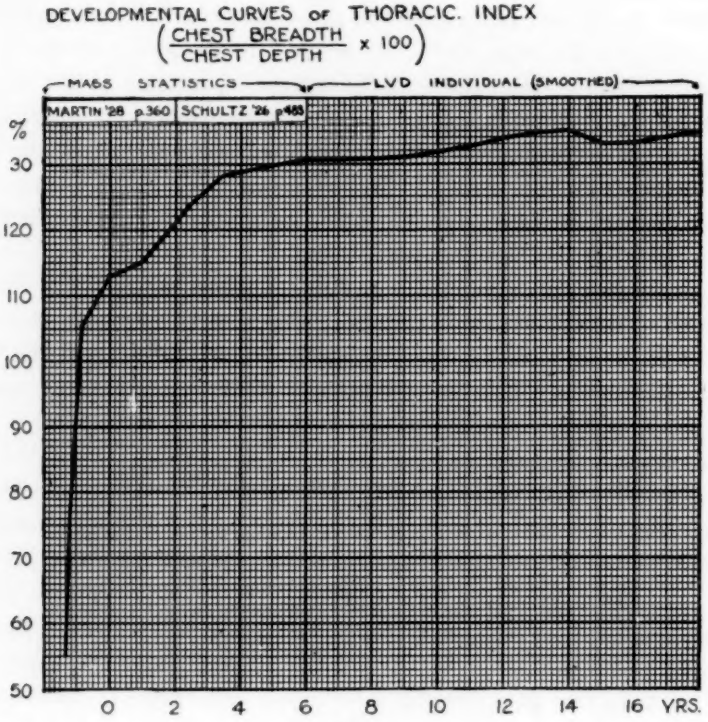

FIG. 2

TABLE 1

Age Changes in Thoracic Index

Foetal and Neonatal

APPROXIMATE AGE	1 MO.	1½ MO.	2 MO.	2½ MO.	3 MO.	4 MO.	5 MO.	8 MO.	NEWBORN
Bradford-Rhodes; Martin, '28, p. 360	55.1	66.6	78	105	110	105	105	111	113.6

APPROXIMATE AGE	9TH FETAL MONTH	10TH F. M.	FULL TERM	1-3 DAYS POSTNATAL	5TH DAY	7TH DAY	9-10TH DAY
Scammon & Rucker, '21	115.6	116.3	116.3	98.0	99.5	98.0	99.5
Schultz, '26, p. 483	118.5	....	....	....	....	....	....

Postnatal

APPROXIMATE AGE		1ST YR.	1-5 YR.	6-10	11-15	16-20	21-25	26-30	31-40	41-50	51-60	61-70	OVER 70
White, Martin, '28	♀	....	....	....	....	136.7	132.9	....	....	....	....	....	....
White, Martin, '28	♂	....	....	132.8	138.4	....	134.0	....	135.8	....	....	....	129.2
White, Sack	♂	....	....	130	132	....	....	....	....	....	....	....	....
White, Hrdlička, '00	♂	....	....	140	141	....	....	....	....	....	....	....	....
White, Hoesch-Ernst, '06	♂	....	....	136	140	....	....	....	....	....	....	....	....
White, Rouma, '21	..	....	....	139	137	....	....	....	....	....	....	....	....
Negro, Rouma, '21	..	....	....	138	140	....	....	....	....	....	....	....	....
Negro, Rodes, '06	♂	....	132.4	137.8	140.2	140.4	142.5	143.5	139.3	137.4	136.9	135.8	138.4
Negro, Rodes, '06	♀	116.5	130.1	137.0	145.5	142.1	139.7	139.7	138.0	134.3	133.5	132.9	130.4

The form of the chest is modifiable by exercise. Veterans of the World War returning to the United States had broad and shallow chests —135.5 in whites, 137 in negroes. Athletes engaged in the heavier types of contests, particularly wrestling, gain exceptionally broad shoulders (Martin, '25, Fig. p. 43). The fact that the chest index responds to exercise of the chest does not, of course, diminish the importance of the genetical factors in producing the various types of chest index.

3. RESULTS

1. Mass Statistics

The thoracic index of BOA Nordic boys tends, in our series and with our technique, *on the average,* to rise from about 130.5 per cent at 8 years to about 135 at 12 years, or about 1.1 point per year. It thereupon decreases to about 130 per cent at 16 years. Here, as we may infer from measurements of older boys, comes another turning point and the index rises smartly; i.e., as maturity is gained the chest broadens markedly. (Fig. 3; LVD ♂ curve.) This changing index tells us that

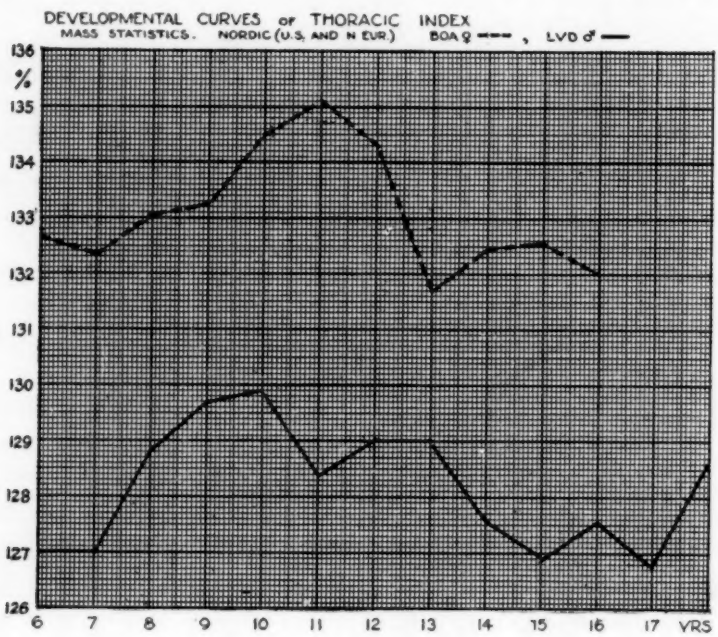

FIG. 3

in the male, on the average, the transverse diameter of the chest (which was only 16 per cent greater than the depth at birth) increases until the early juvenile chest breadth is about 1.30 times the depth; while the adolescent chest is about 1.35 times the depth. Thereafter a temporary relative decrease occurs until chest breadth has fallen, as puberty sets in, to 1.30 times depth. But in the postpubertal period, 16 years onward, the chest probably broadens relatively till its breadth is again 1.35 times depth. The curve of LVD boys (Fig. 3) lies at a lower level than that of BOA boys; but shows the preadolescent maximum, with the prepubertal sag, followed by the temporary pubertal increase of chest depth.

If my ratios are slightly less than those obtained by Hoesch-Ernst for Zurich boys, that may be due in part to a difference of technique by which our mean, intertangential, chest depth is greater than hers (xiphosternal-vertebral). On the other hand our mean index at adolescence agrees pretty closely with the European index as given by Martin ('28, p. 361).

Sexual. In our BOA Nordic girls the chest index rises, on the average, from about 132 at 6 years of age, to 135 at 11 years, and then falls slightly to about 132 at 13 years. It thereafter rises slightly (Fig. 3). This precocity of the girl over the boy in attaining the maximum prepubertal eccentricity of thoracic cross-section is striking. It is found not only in our data; it appears in those of Hoesch-Ernst ('06, Tab. 15) as follows:

AGE	8-9	9-10	10-11	11-12	12-13	13-14	14-15 YEARS
Boys	139	134	138	138	138	141	142
Girls	139	140	138	146	143	140	141

Here the chest attains its greatest relative breadth in the average girl at 11 years; in the average boy at 12-15 years. This is thus another example of female precocity in growth, found also in stature and many other dimensions.

Social. While the curve of the mean of the thoracic index of the BOA and LVD boys runs fairly close to the mean of all our boys, the mean of the mongoloid dwarfs finds its maximum of about 140 at 19 years of age. This group seems to have exceptionally elliptical chests.

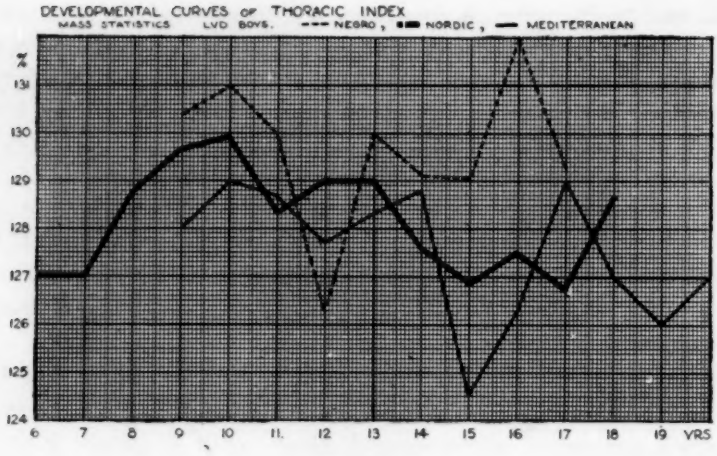

FIG. 4

Racial. As Figure 4 shows, the average thoracic index for Nordic LVD boys has a relatively high value between 8 and 14 years (around 129 and 130), then comes the decline of 3 points at 15 and 16 years, followed by a rise again. In the Mediterraneans of our series the thoracic index reaches a prepubertal maximum of only 129. The American "Negro" children seem, on the whole, to have, at 16 years, an index (of 132 or more) which is larger than that of European stock.

In adult male Old Americans, Hrdlička ('25, p. 308) finds an index of 137, to be compared with the index of 134.5 for white males demobilized in 1919 (Davenport and Love, 1921, p. 209) and with Hrdlička's ('00) asylum children of 16 years, 146.

The adult North American Indians show, on the average, broad chest; 138 to 144 (Martin, '28, p. 362). This may be associated with their outdoor life. Papillault ('06, p. 67) found for 3 "Americans" an index of 159! However, for the Maya Indians Steggerda ('32) gets an adult index of only 134; which is about the same as demobilized U. S. soldiers. Papillault (1906) held that the Negro possesses a somewhat rounder type of thorax than Europeans; finding indices of 138 to 145. However, with an index of 138, according to Martin ('28, p. 362) the "Afrikanischer Neger" stands high. Rodes (1906) finds for the adult "American Negro" male thoracic indices up to 143. In the case of U. S. troops measured at demobilization, 1919, the index found was for whites

134.5; for "Negroes" 137. Accordingly it would appear that the Negro has, on the average, a somewhat higher eccentricity of the chest ellipse than the white.

2. *Individuals*

The simplest type of curve of the development of the thoracic index exhibits, within the given age range, two maxima and an intervening minimum. Some individual examples of this type are shown in Figure 5. In M. H., T. F., and J. K. there is an adolescent maximum at

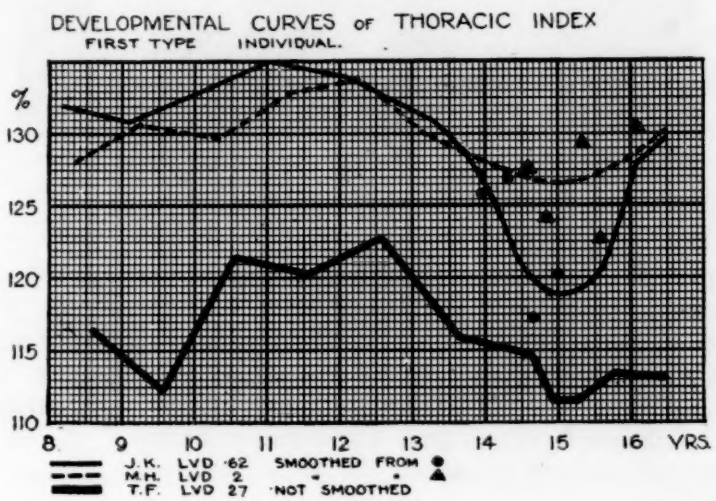

FIG. 5

about 12 years, a minimum at 15 and a rise to a postpubertal maximum thereafter. The height of the several curves on the chart is various, indicating that these three boys differ in the shape of the thorax at nearly all ages.

A second type of developmental curve is even commoner (Fig. 6). This is characterized by two or three maxima and two minima between the ages of 8 and 18 years. Of the maxima the main one is in late adolescence, between 13 and 14 years; one is in the late juvenile stage, about 10 or 11 years, and a third maximum corresponds with the second of the other type—late pubertal. The difference between the two types

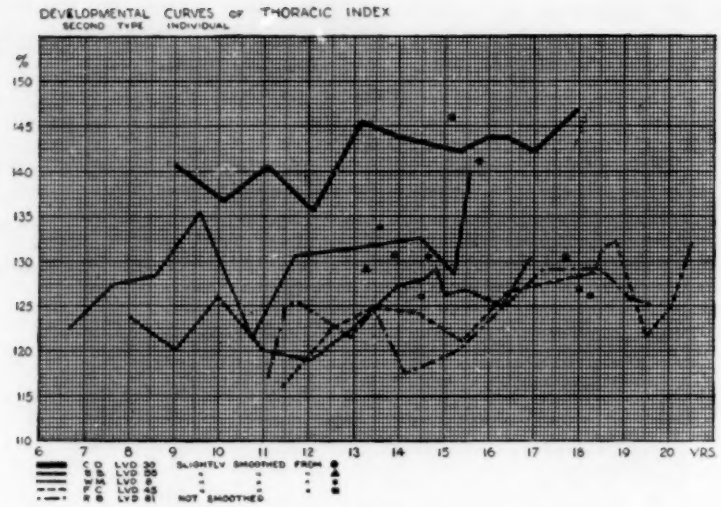

FIG. 6

is apparently the replacement of the adolescent maximum of type 1 by two maxima separated by a depression at 11 or 12 years.

In general, the thoracic index (chest breadth in terms of depth) tends to increase from 6 to 18 years, altogether about 6 points in 12 years. The course of this increase is, however, clearly not rectilinear; the chest breadth tends to grow faster than depth for a year or two during early puberty, sometimes also before adolescence sets in; and again at late puberty.

The changes in the thoracic index during development clearly depend upon changes in the chest breadth and the chest depth, and the relation between them. This relation may be illustrated by the case of J. K. As Figure 5 shows, his thoracic index fell slightly from 8 to 9 years, rose to a maximum at 11 years, then declined to 15 years. Thereafter it rose for a time. Figure 7 shows for the same J. K. the growth of chest breadth and chest depth. The curves approach each other at 15 years (low index). Figure 8 shows the approximate cross-section of J. K.'s chest at three ages. Figure 7 shows, for comparison, the growth of his stature.

Figure 9 shows for a number of boys the growth curves of chest breadth and chest depth, (the latter smoothed slightly at ages 15-16).

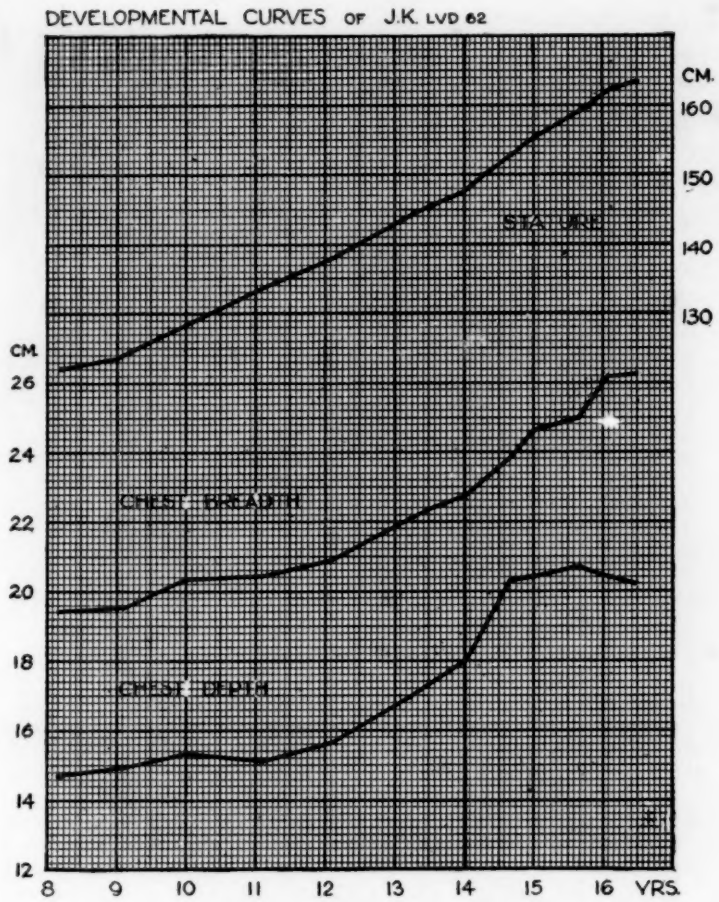

FIG. 7

The nearer together the curves approach, the nearer the thoracic index to 100. One sees that the two curves are concave toward each other at around -2 to -3 years and farthest apart at $+2$ to $+3$ of the pubertal period. They approach most closely usually at about 15 years, (point of centering), due to the more rapid growth of chest depth for a year or two, then chest breadth grows the more rapidly and the thoracic index begins to increase again. In general the increase of thoracic index at the preadolescent period seems to be due to a temporary rapid growth of chest breadth and a slow growth of chest depth at that period.

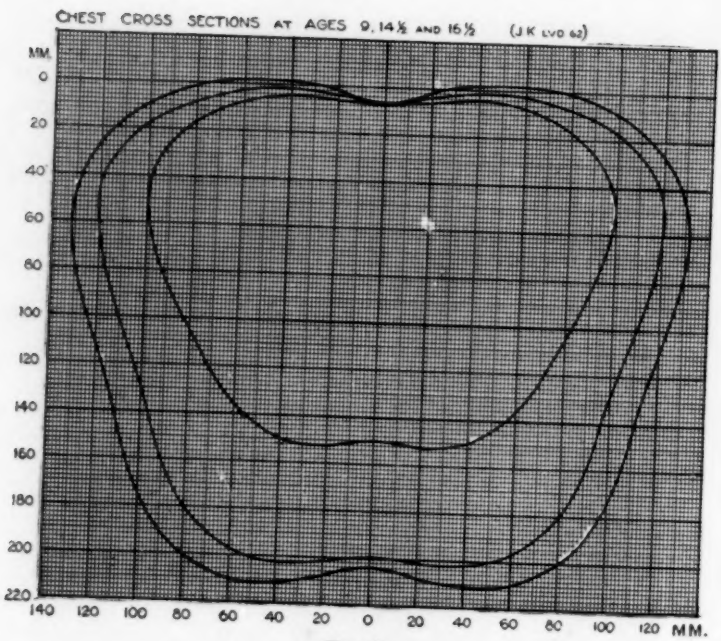
CHEST CROSS SECTIONS AT AGES 9, 14½ AND 16½ (J.K LVD 62)
MM.
0
20
40
60
80
100
120
140
160
180
200
220
140 120 100 80 60 40 20 0 20 40 60 80 100 120 MM.

FIG. 8

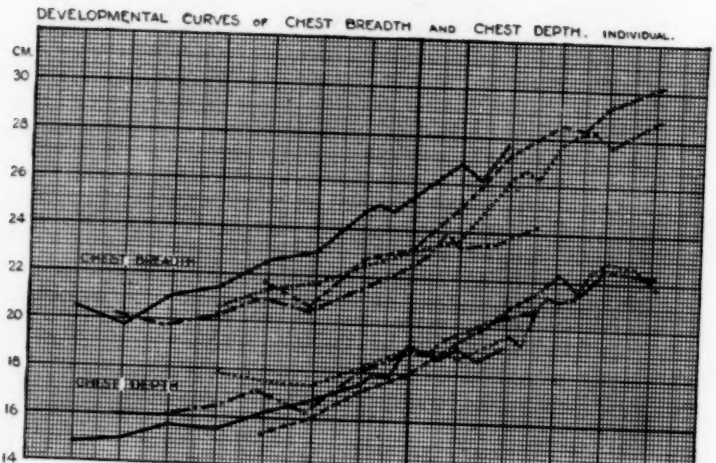
DEVELOPMENTAL CURVES OF CHEST BREADTH AND CHEST DEPTH. INDIVIDUAL.
CM.
30
28
26
24
22
20
18
16
14
CHEST BREADTH
CHEST DEPTH
-8 -7 -6 -5 -4 -3 -2 -1 0 1 2 3 4 5 YRS.
CENTERED AT AGE

FIG. 9

Typically, chest breadth increases more slowly at the early adolescent period than in the years immediately previous and following. If we use sitting height of the suprasternal notch (front sitting trunk length) as a reference distance; and if we compare the ratio of transverse chest to this sitting trunk length at different ages; then we get the result shown in Figure 10. This figure gives for six boys the changing trunk

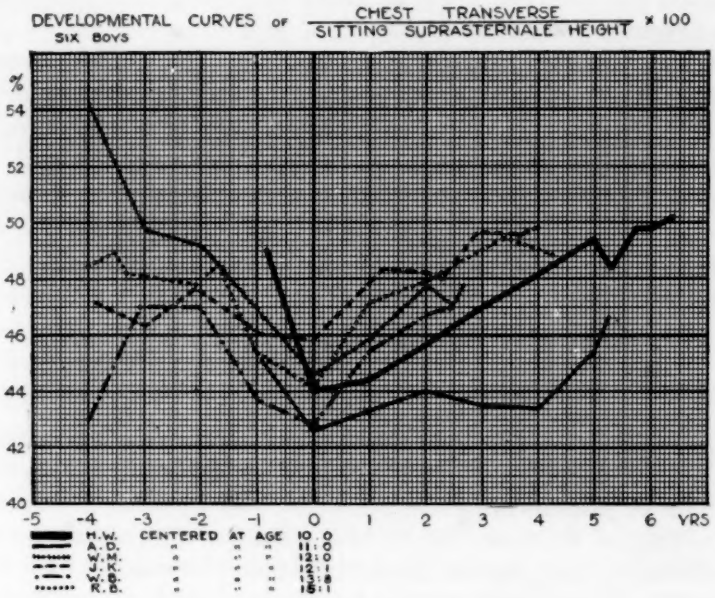

FIG. 10

breadth-length index. The curves are referred to an abscissal point which is not that of the same absolute age but of the adolescent minimum. To the right and left of this reference point are the indices in successive years before and after this reference point. The age of this minimum varies in the six boys from 10:1 to 15:1. The minimum ratio varies from 42.7 per cent to 45.9 per cent. At 4 years before the minimum the ratio averages about 48 per cent; at 4 years after, about 47.5 per cent.

If we compare the ratio of chest depth to sitting trunk length at different ages we get results that are in marked contrast to Figure 10.

The curves are more nearly level. A very slight depression occurs, but this is at a year or two later than the depression of the chest breadth ÷ sitting suprasternale; and combined with an increasing chest breadth index at this time tends to create that increased thoracic index found at about 16 years. It is rather remarkable and hitherto apparently overlooked, that the chest depth is so much more conservative in growth, grows so much more closely with trunk growth as a whole, than chest breadth. For the latter grows for a time more slowly than trunk length and then more rapidly.

To sum up: The thoracic index tends to increase from birth to maturity but an individual undergoes many fluctuations in this ratio. In general the increment is interrupted by one or more temporary decreases of which the most marked occurs in boys at about 14-15 years of age. At this temporary decrease the thoracic index may fall 5 to 10 points, for example, from 126 to 119 per cent. This interruption is due to a remarkable relative slowing up of the process of increasing chest breadth.

4. DISCUSSION

PHYLOGENY OF MAN'S THORACIC INDEX

Admitting that man's adult thoracic index of around 135 per cent is an adaptive one, the question still remains to be considered: How did man come by just this index?

The topic of the reason for the broad, shallow chest of man as contrasted with the deep, narrow chest of the cow (for example) has been discussed by many investigators and different views have been held by them.

Wiedersheim ('08) recognized two types of thorax among mammals: Type A in which the index is less than 100 and Type B in which it exceeds 100. He says type A is found in ruminants, pachyderms and carnivores; type B in Marsupialia, beaver and other rodents, insectivores, Cetacea, bats, otters, anthropoids and man. He concludes that type A is phylogenetically older than type B and he says that in the human embryo the cephalad portion of the thorax shows type A but this is modified in the caudad portion. He thinks the passage from the embryonic deep chest to the adult flat chest is a recapitulation of the phylogenetic series, starting from a quadruped form in which the thorax is suspended beneath the spine; ending in erect man.

Schultz ('26) criticizes this interpretation, calling attention to the fact that in the fetal dog the chest is of type B, which could hardly have been an ancestral type. "The chest type in early embryonic life can not be regarded as the onto-

genic reappearance of some phylogenetic condition, but is directly determined by causes of a purely topographic nature, especially by the growth of heart, lungs, and liver. After birth gravity is the most potent factor influencing the thorax shape. In quadrupeds the weight of the organs exerts a constant pressure on the sternum, thereby tending to increase the depth of the chest. In man the weight of the organs acts in a different direction, i.e., on the diaphragm. Finally, in the sloth, which habitually assumes a more or less upright position or hangs below a horizontal limb of a tree suspended by all four appendages, gravity affects the chest in just the opposite direction as in quadrupeds, producing a chest index of 160, in contrast to one of about 72 in the adult dog," and Schultz shows that while the fetal sloth has an index of about 100, in postnatal life this becomes 160. Jackson (1907) measured the thoracic index of a puppy at birth and found it about 89, in another dog from the same litter this had decreased in adult life to 74.5. In a third dog of this litter, deprived of both forelegs and forced to assume the upright position for 12 hours daily during one year, the index during this time remained practically constant at about 87. Though the chest did not become as deep as in the control, it certainly did not become more nearly circular on cross-section.

To get a better perspective on this matter I have measured the transverse and sagittal diameters of the thorax at about the sixth rib in a number of mounted skeletons in the American Museum of Natural History, New York City.*

The measuring was done by the aid of three calipers; the Martin anthropometer used as calipers, the sliding calipers of Hermann, and a pelvimeter. It was early seen that the sagittal diameter measured as in the living, from the mesosternum to the tip of the 4th or 5th vertebral spine was, in certain mammals, like the bison, greatly exaggerated. This is owing to the great length of the spine developed for attachment of the nuchal ligament, which is of huge size in order to carry the heavy head. To eliminate the spine, measurement was made from the periferal surface of the mesosternum (at sixth rib) to the ventral head of the rib at the sixth thoracic vertebra. The thoracic index is computed from both measurements. Of course measurements taken in this way on articulated skeletons are subject to an error (probably slight) due to the technique of mounting. Also the indices are only comparable among themselves and cannot be closely compared with measurements taken on the living.

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* The assistance in this undertaking of Mr. H. E. Anthony, Curator, and Mr. Goodwin of the museum staff is gratefully acknowledged. Mr. William Drager acted as recorder and computed the indices.

Later, with the kind assistance of Mr. Gerrit S. Miller, Jr., curator of mammals of the U. S. National Museum, I measured a number of "thoracic baskets" of mammals in the study collections of the museum, and also a number of mounted articulated skeletons.

There is doubtless a considerable error in relying upon such skeletal material. I have the impression that the articulated skeletons preserve more nearly the living proportions than the dead thoracic baskets, especially if the latter belong to fairly large species. The exigencies of space in packing for transit to the museum, and even of drawer space in the study series of the museum may have caused some distortion. Accordingly I have used only data obtained from two or more measurements of the same species, except for certain ratios which have been enclosed in brackets. Weisgerber, also, has reached the conclusion that skeletal material can be used with caution, as a substitute for the living animals.

In some ways the measurements on the skeletons are more significant than those on the living mammals; for, as stated, the latter dimensions are greatly complicated by the great diversity of length of the vertebral spines. This spine length has little to do with the thorax in the strict sense.

Weisgerber gives an exterior and interior measurement of the skeletons studied by him. Although he does not define precisely his technique, I infer that his interior measurement is rather close to mine. He may, however, have measured to the visceral aspect of the sternum (instead of the dermal aspect) in which case his indices would tend to be slightly greater than mine.

The measurements made, averaged and grouped, are given in Table 2. The table includes some data from Weisgerber.

What conclusions can be drawn from a study of this list?

First, there is a rough relation between taxonomic position and thoracic index. The most primitive mammals stand at or near the upper end of the list as though a broad thorax was a primitive trait of mammals. The ruminants are found at the bottom of the list. The primates occupy an intermediate position, and, of these, the anthropoids a high one. Most Insectivora and all bats occur in the upper part of the list; likewise most Edentata.

Second, the taxonomic position is of importance only if the habits of the order are fairly uniform. Thus the Marsupialia, though a primitive group, vary greatly in habits and correspondingly in thoracic

TABLE 2

TABLE 2

The Thoracic Index of Various Mammals Arranged in Order of Size of Index and Classified Taxonomically and by Habits. The Numbers in Parentheses are from Weisgerber ('79)

	THORACIC INDEX			PRIMITIVES	BURROWERS	FLIERS	ARMORED	SWIMMERS	RUNNERS	TERRESTRIAL WALKERS AND JUMPERS	ARBOREAL
	New data	Weisgerber									
Ornithorhynchus	169	(153)	Monotreme	X	..	..	..	..	..	..	..
Manatus	169	.....	Sirenia	..	..	..	..	X	..	..	..
Centetidæ	164	.....	Insectiv.: earthworm eater	..	X	..	..	..	..	..	..
Scalopus, et al.	162	(159)	Mole	..	X	..	..	..	..	..	..
Erethizon	157	.....	Porcupine	..	..	..	..	..	..	X	..
Echidna	155	(149)	Monotreme	X	..	..	..	..	..	..	..
Notoryctidæ	155	.....	Marsupial "mole"	..	X	..	..	..	..	..	..
Molossidæ	149	.....	Bats	..	..	X	..	..	..	..	..
Homo sapiens	148	(160)	European	..	..	..	..	..	..	X	..
Dasypus	146	.....	Armadillo	..	..	..	X	..	..	..	..
Gorilla	142	(141*)	* juvenile	..	..	..	..	..	..	X	X
Chimpanzee	141	(158*)	* juvenile	..	..	..	..	..	..	..	X
Orang utan	140	(132)		..	..	..	..	..	..	..	X
Soricidæ	139	.....	Shrews	..	X	..	..	..	..	X	..
Gibbon	138	(131)		..	..	..	..	..	..	..	X
Ursus	138	.....	Carniv.: Bear	..	..	..	..	..	..	X	..
Viscaccia	136	.....	Rodent: burrowing	..	X	..	..	..	..	..	..
Rhinocerotidæ	135	.....	Rhinoceros	..	..	..	..	..	..	X	..
Phalangeridæ, flying spec.	133	.....	Marsupialia	..	..	X	..	..	..	..	..
Bradypus	130	(130*)	* "Edentates"	..	..	..	..	..	..	..	X
Cynopterus	129	.....	Bats	..	..	X	..	..	..	..	..

TABLE 2—*Continued*

The Thoracic Index of Various Mammals Arranged in Order of Size of Index and Classified Taxonomically and by Habits. The Numbers in Parentheses are from Weisgerber ('79)

	THORACIC INDEX			PRIMITIVES	BURROWERS	FLIERS	ARMORED	SWIMMERS	RUNNERS	TERRESTRIAL WALKERS AND JUMPERS	ARBOREAL
	New data	Weis-gerber									
Cyclopes and *Tamandua*	129	.....	Edentates, arbor.	..	..	..	..	..	..	..	X
Manis	128	.....	Edentates, pangolin	..	..	..	X	..	..	..	..
Hapalidæ	127	.....	New World monkeys	..	..	..	..	..	..	..	X
Perodipus	127	.....	Kangaroo rats	..	..	..	..	..	..	X	..
Eutamias and *Petaurista*	126	.....	Rodentia: squirrels	..	..	..	..	..	..	X	X
Phascolarctinæ	126	.....	Marsup.: sloth-like	..	..	..	..	..	..	..	X
Pteropus	124	.....	Bats: "flying fox"	..	..	X	..	..	..	..	..
Halicore	123	.....	Dugong	..	..	..	..	X	..	..	..
Erinaceus	123	(159)	Insectiv.: spiny	..	X	..	..	..	..	X	..
Tarsius	122	.....	Tarsiidae	..	..	..	..	..	..	..	X
Cholæpus	121	.....	Sloth	..	..	..	..	..	..	..	X
Ondatra	121	.....	Muskrat	..	X	..	..	X	..	..	..
Phyllostomatidæ	121	.....	Leaf nosed bats	..	..	X	..	..	..	..	..
Mustelidæ	120	.....	Weasels, etc.	..	X	..	..	X	..	..	..
Phalangeridæ, non-flying species	120	.....	Marsupial	..	..	..	..	..	..	X	..
Caviidæ	120	.....	Guinea pigs	..	..	..	..	..	..	X	..
Noctilionidæ	120	.....	Bats	..	..	X	..	..	..	..	..
Macropus	119	.....	Kangaroo	..	..	..	..	..	..	X	..
Lorisinæ	116	(106)	Lorises	..	..	..	..	..	..	..	X
Orycteropus	115	.....	Cape anteater	..	..	..	..	..	..	X	..
Pithecidæ	111	(120)	Monkeys	..	..	..	..	..	..	..	X
Chiromyidæ	110	.....	Lemur: Aye-aye	..	..	..	..	..	..	..	X

TABLE 2—*Continued*

TABLE 2—*Continued*

Pygathrix	109	(117)	Old World Monkeys	..	..	..	..	..	..	..	X
Lagothrix	109	.....		..	..	..	..	..	..	..	X
Myrmecophaga	109	.....	Anteater	..	..	..	..	..	..	X	..
Solenodon	108	.....	Habits little known	..	X	..	..	..	..	..	..
Lemur	107	.....	Lemuroidea	..	..	..	..	..	..	..	X
Papio	107	.....	Baboon	..	..	..	..	..	..	..	X
Hystricidæ	107	.....	European porcup.	..	..	..	..	..	..	X	..
Cercopithecus	106	.....	Old World monkey	..	..	..	..	..	..	..	X
Prodelphinus	105	.....	Dolphin	..	..	..	..	X	..	..	..
Peramelidæ	102	.....	Marsup.: rat-like	..	X	..	..	..	..	X	..
Canidæ	102	(98)	Dogs	..	..	..	..	..	X	..	..
Macacus	102	(97)	Monkeys	..	..	..	..	..	..	..	X
Presbytis	102	(106)	Lemuroidea	..	..	..	..	..	..	..	X
Neomeris and *Phocaena*	99	(107)	Cetacea	..	..	..	..	X	..	..	..
Zalophus	99	.....	Sea Lion	..	..	..	..	X	..	..	..
Leporidæ	96	.....	Hare and rabbit	..	..	..	..	..	..	X	..
Chrysothrix	96	.....	Old World monkey	..	..	..	..	..	..	..	X
Saimiri	96	.....	Old World monkey	..	..	..	..	..	..	..	X
Hyracoidea	95	.....	*Hyrax*, coney	..	..	..	..	..	..	X	..
Sciurus	94	.....	Squirrel	..	..	..	..	..	..	X	X
Callorhinus	91	.....	Fur seal	..	..	..	..	X	..	..	..
Lynx	91	.....	Wild cat	..	..	..	..	..	X	..	..
Tupaia	89	.....	Insectiv.: squirrel-like	..	..	..	..	..	..	..	X
Viverrinæ	89	.....	Civet cats	..	..	..	..	..	X	..	..
Bison	86	.....		..	..	..	..	..	X	..	..
Herpestinæ	86	.....	Viverridæ	..	..	..	..	..	X	..	..
Elephas	82	(98)	Elephant	..	..	..	..	..	..	X	..
Felis	81	(100)	Cats	..	..	..	..	..	X	..	..
Antelopes and Giraffe	75	(75)		..	..	..	..	..	X	..	..

index. For example, the Notoryctidæ, or marsupial moles, have a high index, 155; while the Peramelidæ, or marsupial rats, have a much lower index, 102. Indeed, in the same marsupial family of Phalangeridæ the flying species have an average index of 133; the non-flying species an index of 120.

Third, the mammals that have similar habitat tend in some cases to have a somewhat similar thoracic index. Thus the burrowers have a rather high index whether Insectivora like the Centetidæ, 164, or marsupials, like the Notoryctidæ, 155; or a rodent, like *Viscaccia,* 136; or a carnivore, like the otter, 120. The swift runners have a low index whether ruminants like the antelope, giraffe and bison, or carnivora, like cats. The fliers have a rather high index whether bats, flying marsupials (Phalangers, 133), or flying squirrels (of which one specimen, not listed, gave an index of 173, which was doubtless too large) (See Fig. 11)

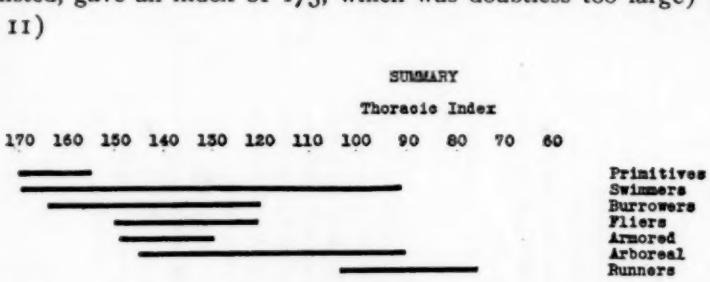

FIG. 11. RANGE OF AVERAGE THORACIC INDICES OF ADULT MAMMALS HAVING DIFFERENT TYPES OF HABITAT AS INDICATED AT RIGHT OF FIGURE

Fourth, the view that narrowness of the chest is correlated with absence of clavicle has certain elements of truth. The running mammals, such as the hoofed ungulates, have indeed no clavicle; also in most dogs and the cats the clavicle is rudimentary. However, the bear, with a broad chest has no clavicle, nor has *Manis,* the anteater (Flower, '85, pp. 255, 259). The correlation is not very high; one cannot say the chest is narrow because there are no clavicles.

Fifth, the end result is clearly a combination of endogenous (genetic) and exogenous factors, as we see in the case of the Phalangers. Nevertheless the genetic factor seems the stronger. Thus the Edentata all are found to have broad chests, whether armored—like the terrestrial armadillo (146); unarmored, anteating—like *Orycteropus* (115); or un-

armored arboreal, whether hanging horizontally—like *Bradypus* (130), or not—like *Tamandua* (129).

It follows from the foregoing considerations that one is not justified in concluding because the sloth has a broad chest, that broadness is merely the result of gravity; for *Bradypus* belongs to a broad-chested order, whether the representatives live in trees or on the ground. Indeed, one may say that the broad chest of *Bradypus* enables it to hang from a large limb in a way which would be tiresome or difficult for an animal with narrow chest. The form of chest has made possible—as it were, suggested—the habitat; quite as probably as the reverse.

Returning to man, in the form of his chest he agrees with the monkeys in general in not having a narrow chest, and with the anthropoids in having a broad chest, a condition that reaches an extreme in the adult gorilla which does not walk erect, but only at a considerable angle. Similarly, gravity is not the sole cause of the deep chest of the gazelle. *Echidna,* which walks on the ground has a chest shallower than that of man and the bear has a round chest close to that of the anthropoids in form. But *Echidna* and the bear are not graceful runners. One may conclude that it is no less true that the gazelle is a graceful runner because its chest is narrow than vice versa.

SUMMARY

The measurements of the chief diameters of the thorax in growing children show that the thorax undergoes certain marked changes in proportions. The thoracic index increases on the average to about 14 years of age (with some fluctuations), decreases usually at about 15 years, and then increases again. This temporary decrease occurs at the period of rapid growth, when the trunk is elongating rapidly. At this time the chest breadth is growing less rapidly than other dimensions of the trunk and the chest becomes more nearly a circular cylinder. But as growth of other dimensions of the trunk slacks up the breadth continues to increase and the adult proportions of chest breadth, 130 to 145 per cent, are achieved.

In girls the maximum eccentricity of chest cross-section is achieved a year or two earlier than in boys; corresponding to the earlier adolescence of girls. The American Negroes, children and adults, have a somewhat more flattened cross-section than whites.

The broad, shallow chest of man is not to be ascribed wholly to his upright position. The anthropoid apes all have elliptical chests, although none of them habitually walks erect; man belongs to a broad chested genetic group. This shape does not interfere with the upright posture. Gravity may have an influence in modifying it. But selection also plays a rôle in securing fitness of the species for conditions of life.

In mammals there is a rough relation between the taxonomic position and the size of the thoracic index.

The taxonomic position is of importance only if the habits of the order are fairly uniform.

Mammals that have a similar habitat even though they belong to different orders tend in some cases to have a somewhat similar thoracic index (adaptation to the same environment).

The correlation between narrowness of the chest and absence of clavicle is not very high.

The end result, thoracic index, is a combination of endogenous (genetic) and exogenous (environmental) factors. The genetical factors are apparently the stronger. Environment acts probably chiefly as selector, but gravity may act directly.

The narrow-chested running mammals are not so much narrow-chested because runners, as runners because narrow-chested.

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A NOTE ON THE PROBLEM OF MIRROR-IMAGING IN HUMAN TWINS

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IT IS sometimes assumed that monozygotic or identical twins may be compared to two symmetrical halves of a single person. This notion has led certain investigators on twins such as Wilder (1904), Newman (1917, 1923, 1928), and Dahlberg (1926) to seek for a mirror-imaging tendency between two individuals of the same twin pair, especially with respect to handedness, footedness, the direction of hair-whorl, friction ridges of fingers, palms and soles, as well as various anatomical and pathological features. But certain other authors like v. Verschuer (1928, 1932), Obonai (1930), and White and Jones (1932) have arrived at conclusions which are at variance with this idea at least concerning handedness and hair-whorl. The present study was undertaken to contribute something to this disputed question; and the results obtained for handedness and hair-whorl are given in this paper.

The twins employed as material for the study consist of 95 male pairs, 68 female pairs and 17 different-sex pairs found among primary-school children of Osaka. The zygosity of the same-sex twins was diagnosed on as many criteria as were available, such as the physiognomy, finger-, palm- and sole-prints, blood-type, ear-form, eye- and hair-color, and anthropological and psychological measurements. Thus the same-sex twins have been classified into 118 monozygotic pairs consisting of 70 male and 48 female pairs, and 45 dizygotic pairs of which 25 are male and 20 are female pairs.[1] There are a few cases where the diagnosis of zygosity is somewhat uncertain. But the number of such cases is so small that the conclusions to be presented below would not be affected whether these twins are included in the monozygotic or dizygotic group.

[1] Among Japanese, monozygotic twins are much commoner than dizygotic as pointed out in another paper of the authors, 1933.

HANDEDNESS AND FOOTEDNESS

The handedness and footedness of these twins were examined with reference to the same criteria which we had employed for diagnosing the handedness and footedness of single-born school children.[2] But, instead of using the questionnaire method, each child was given a ball, for instance, and asked to show which hand he threw it with. Similar methods were employed for all the other criteria. The results are presented in Table 1, together with the values obtained from single-born

TABLE 1

Frequency of Left-handedness and Left-footedness in Twins as Compared With That in Single-born Individuals, According to Each Criterion

	SEX	NUMBER OF INDIVIDUALS	PERCENTAGES OF LEFT-HANDED OR LEFT-FOOTED						
			Penknife	Ball	Scissors	Match	Chopsticks	Writing-brush	Football
Single-born	♂	8718	6.8	7.1	5.7	4.9	2.1	1.1	6.6
	♀	7549	5.1	4.7	3.4	3.1	1.6	1.2	4.7
Monozygotic twins	♂	140	3.6	6.4	2.9	5.0	3.6	2.9	11.4
	♀	96	7.3	7.3	4.2	4.2	2.1	2.1	9.4
Dizygotic twins	♂	67	11.8	11.8	14.9	10.4	4.5	4.5	11.8
	♀	57	10.5	7.0	5.3	5.3	1.8	1.8	10.5

individuals. The frequency of left-handedness and left-footedness is somewhat higher in twins than in single-borns, except for a few special cases which are probably to be attributed to mere chance. This is especially true of the dizygotic group. The same tendency may be perceived in the case where all the individuals who use their left hands in any one of those works are diagnosed as left-handers, as shown in Table 2.

TABLE 2

Frequency of Left-handedness in Twins as Compared With That in Single-born Individuals, According to All Criteria

AGE	TWINS					SINGLE-BORN	
	Monozygotic		Dizygotic				
	♂ ♂	♀ ♀	♂ ♂	♀ ♀	♂ ♀	♂	♀
7-10	11.1	14.8	25.0	15.0	33.3	12.3	10.3
11-14	8.3	11.9	17.8	5.0	15.0	10.7	7.6

[2] See the other paper by the same authors published in HUMAN BIOLOGY.

TABLE 3

Frequency of Different Combinations of Handedness and Footedness Among Twin-pairs According to Each Criterion

	SEX	NUMBER OF PAIRS	CRITERION	PENKNIFE			BALL			SCISSORS			MATCH			CHOPSTICKS			WRITING-BRUSH			FOOTBALL		
			Combination	RR	RL	LL	RR	RL	LL	RR	RL	LL	RR	RL	LL	RR	RL	LL	RR	RL	LL	RR	RL	LL
Monozygotic	♂♂	70	Number	65	5	0	61	9	0	66	4	0	63	7	0	65	5	0	66	4	0	57	10	3
			Obsd. ratio	92.9	7.1	0.0	87.1	12.9	0.0	94.4	5.6	0.0	90.0	10.0	0.0	92.9	7.1	0.0	94.4	5.6	0.0	81.4	14.3	4.3
			Expd. ratio	92.9	7.0	0.1	87.6	12.0	0.4	94.3	5.6	0.1	90.2	9.5	0.3	92.9	7.0	0.1	94.3	5.6	0.1	78.5	20.2	1.3
	♀♀	48	Number	41	7	0	41	7	0	44	4	0	44	4	0	46	2	0	46	2	0	40	7	1
			Obsd. ratio	85.4	14.6	0.0	85.4	14.6	0.0	91.7	8.3	0.0	91.7	8.3	0.0	95.9	4.1	0.0	95.9	4.1	0.0	83.3	14.6	2.1
			Expd. ratio	86.0	13.5	0.5	86.0	13.5	0.5	91.8	8.0	0.2	91.8	8.0	0.2	95.6	4.0	0.4	95.6	2.0	0.4	82.1	17.0	0.9
Dizygotic	♂♂	25	Number	21	3	1	19	5	1	18	6	1	21	3	1	24	0	1	24	0	1	18	7	0
			Obsd. ratio	84.0	12.0	4.0	76.0	20.0	4.0	72.0	24.0	4.0	84.0	12.0	4.0	96.0	0.0	4.0	96.0	0.0	4.0	72.0	28.0	0.0
			Expd. ratio	81.0	18.0	1.0	74.0	24.0	2.0	70.6	26.8	2.6	81.0	18.0	1.0	88.3	11.3	0.4	88.3	11.3	0.4	74.0	24.0	2.0
	♀♀	20	Number	16	4	0	18	2	0	18	2	0	19	1	0	20	0	0	20	0	0	18	2	0
			Obsd. ratio	80.0	20.0	0.0	90.0	10.0	0.0	90.0	10.0	0.0	95.0	5.0	0.0	100.0	0.0	0.0	100.0	0.0	0.0	90.0	10.0	0.0
			Expd. ratio	81.0	18.0	1.0	90.2	9.5	0.3	90.2	9.5	0.3	94.5	4.9	0.6	100.0	0.0	0.0	100.0	0.0	0.0	90.2	9.5	0.3
	♂♀	17	Number	12	5	0	14	3	0	14	3	0	13	4	0	16	1	0	16	1	0	12	5	0
			Obsd. ratio	70.6	29.4	0.0	78.6	21.4	0.0	78.6	21.4	0.0	76.5	23.5	0.0	94.1	5.9	0.0	94.1	5.9	0.0	70.6	29.4	0.0
			Expd. ratio	72.5	25.3	2.2	83.2	16.0	0.8	83.2	16.0	0.8	77.8	20.8	1.4	93.6	5.6	0.8	93.6	5.6	0.8	72.5	25.3	2.2

The fact that the dizygotic group includes more left-handed and left-footed individuals, makes one doubt the above stated idea that the left-handedness or left-footedness of one twin member is to be viewed as due to mirror-imagery.

This problem may also be tested in another way. If the percentage of left-handed children in a given twin population is represented by L and that of right-handed ones by R, then RR: 2RL: LL should be the theoretically expected ratio of the three kinds of combinations which is to be obtained when two individuals among the population are coupled at random. Suppose in the monozygotic twins, handedness is determined entirely by heredity, then the above RL class should become null. But if, on the contrary, the members of every twin pair were to show reversed asymmetry with each other because of some epigenetic influence, then there should be no RR or LL pair. If the ratio is exactly the same as the theoretically expected ratio, there could be neither hereditary nor epigenetic influence, or else the effects of these antagonistic influences would be in a state of equilibrium. Table 3 shows clearly that the observed ratio in every criterion approximates very closely the expected ratio, so that the case seems to conform to the possibility stated last.

Next, let us examine the dizygotic twins concerning this problem. In Table 3, it may be perceived without difficulty that the general tendency is different according to sex of twins. In the female twins the values for RL pairs are near the expected values, while in the male twins they are smaller, and in the different-sex twins they are greater, than the expected values. In short, in the dizygotic twins also, the preponderance of neither hereditary nor epigenetic influence is to be perceived.

If all the criteria are considered simultaneously, we get the values shown in Table 4. Here also, the observed ratio is very close to the

TABLE 4

Relative Frequency of Different Combinations of Handedness in Twin-pairs According to All Criteria

TWIN PAIRS	RATIO	RR %	RL %	LL %
Monozygotic	Observed	80.0	17.5	2.5
	Expected	78.7	20.0	1.3
Dizygotic	Observed	70.0	25.0	5.0
	Expected	68.0	28.0	3.1

expected ratio; moreover, there is no distinction between the monozygotic and dizygotic groups. The general conclusion stated above thus holds true for this case also.

HAIR WHORL

The direction of twisting of the hair whorl of the head is often considered as belonging to the same category as handedness or footedness. The whorl may be of a dextral (clockwise) or sinistral (anticlockwise) type; besides there are comparatively few cases in which there is a double or even triple whorl of various combinations of the twisting directions.

Among 1800 school children of two primary schools in Kyoto, we have found the distribution of the types of the head whorl to be as shown in Table 5. Most Japanese boys of this age have their hair cut

TABLE 5

Relative Frequency of Different Types of Head-whorls in Single-born and Twins

		TOTAL NUMBER OF INDIVIDUALS	DEXTRAL		SINISTRAL		DOUBLE	
			No.	%	No.	%	No.	%
Single-born	♂	963	492	51.1	400	41.5	71	7.4
	♀	837	471	56.3	342	40.9	24	2.4
Twins	Monozygotic	250	145	58.0	86	34.4	19	7.6
	Dizygotic	118	58	49.1	47	40.0	13	11.1

very short, and their whorls are very easy to examine, so that the results obtained from boys are more reliable than those obtained from girls. At any rate, the results in Table 5 seem to show that there is little sexual difference in the distribution of these whorl types. As a rough estimation, it may be said that the dextral whorls are slightly above 50 per cent, the sinistral are about 40 per cent and the double whorls are less than 10 per cent. Obonai (1930) gives 38.7 per cent for the frequency of the sinistral whorl among the children of Tokyo, while Watanabe (1916) has found that about 10 per cent of the children of a primary school of Tokyo have double whorls.

The above estimation is quite different from that of foreign authors, who put the frequency of the sinistral whorl between 18-20 per cent (Lauterbach and Knight, 1927, 18.1 per cent; v. Verschuer, 1932, 18.7-

20 per cent; White and Jones, 1932, 18.9 per cent). The difference is too great to be attributed to mere chance or to the difference in personal judgment. More probably it is a racial distinction. But for a crucial examination of this problem more material from both whites and Japanese may be necessary.

Next, among the twins we studied, the distribution of the whorl-types is as shown also in Table 5. The distribution among both the monozygotic and dizygotic twins is similar to that for single-born individuals. At least there is little evidence that the sinistral whorl is more frequent among twins than among single-born individuals.

Let us also examine how the different types of whorls are distributed among twin-pairs. According to the figures given in Table 5, the R : L ratio for the monozygotic twins is $145 : 86 = 62.8 : 37.2$; consequently, the RR : 2RL : LL ratio is 39.4 : 46.7 : 13.8, while for the dizygotic twins $R : L = 58 : 47 = 55.2 : 44.8$, and RR : 2RL : LL = 30.5 : 49.5 : 20.1. These theoretical values are quite different from the observed values in Table 6. In both types of twins the disharmonious combination RL

TABLE 6

Relative Frequency of Different Combinations of Whorl-types in Twin-pairs

	TOTAL NUMBER OF PAIRS	RR		RL		LL		COMPOSITE *	
		No.	%	No.	%	No.	%	No.	%
Monozygotic	125	58	46.5	21	16.8	30	24.0	16	12.8
Dizygotic	59	18	30.5	16	27.1	15	25.4	10	16.9

* Cases where one or both members have double whorls.

is much smaller in the observed values than in the theoretical. This means that in the determination of the direction of the whorl, heredity plays a rather important rôle. At the same time, the existence of disharmonious combinations even in the monozygotic pairs, speaks for the existence of some epigenetic influence. But from the data we cannot decide whether this epigenetic factor is the mirror-imaging tendency or something else.

But there is another possibility in interpreting the data in Table 6. If the class "Composite" is included in the class RL, as is done by certain previous authors, then the observed ratio of RR : 2RL : LL becomes close to the expected ratio in the dizygotic twins (observed, 30.5 : 44.0 :

25.4; expected, 30.5 : 49.5 : 20.1). In the monozygotic twins, however, the class RL in the observed ratio is still smaller than that in the expected ratio (observed, 46.5 : 29.6 : 24.0; expected, 39.4 : 46.7 : 13.8). The general conclusion just stated, therefore, seems to hold under this circumstance also.

REMARKS

The findings by previous authors on the problem of handedness among twins accord in the point that left-handedness is more commonly met with in the twin population than among single-born individuals. However, they do not agree in the question as to which kind of twins has the higher frequency.

Dahlberg (1927), Newman (1928), Hirsch (1930) and v. Verschuer (1932) got more or less high values in monozygotic twins. Siemens (1924), on the contrary, obtained a higher value in dizygotic twins, while Wilson and Jones' (1932) results show that there is no reliable difference between the two groups, although the values are apparently somewhat higher in the dizygotic group.

The results here obtained likewise seem to favor the dizygotic group.

From the data given above, it seems to be safe to conclude that the reversed asymmetry in handedness and footedness is a phenomenon related somehow to twinning, but that this reversing agency is probably something other than mirror-imaging which is characteristic of monozygotic twinning.

Previous authors' results on the hair whorls of twins are very conflicting.

Newman (1928) found a much higher frequency of the sinistral whorl among monozygotic twins than among dizygotic, whereas Lauterbach's (1925) data show that the sinistral whorl is much commoner among different-sex twins than among same-sex twins. Von Verschuer (1931) obtained a nearly identical value of the frequency of the sinistral whorl for both the monozygotic and dizygotic groups which is somewhat higher than that of single-born individuals. Wilson and Jones (1932) got about the same frequency value in both groups of twins as in single-born individuals.

Our finding stated above agrees with the results obtained by the last-named authors.

In short, there are not sufficient data to show that the sinistral whorl is especially common among twins, nor is there any conclusive evidence that the sinistral whorl is found more frequently among monozygotic twins than among dizygotic. Thus, we have to conclude that the direc-

tion of the hair-whorl has no relation with the twinning phenomenon, and the reversed direction of the whorl is far from being the result of mirror-imaging.

The general conclusion stated above concerning handedness, footedness and twisting direction of the hair-whorl, is supported further by the comparison of the observed RR : RL : LL ratio with the theoretically expected ratio which is to be obtained when two individuals of a given twin population are coupled entirely at random.

SUMMARY

(1) The idea that the reversed asymmetry in handedness, footedness and twisting direction of hair-whorl of monozygotic twins is due to mirror-imaging, was tested by using the material of 118 monozygotic, and 62 dizygotic pairs of Japanese twins.

(2) The handedness was diagnosed on 6 different criteria and the footedness on the determination of the leg used in kicking. The frequency values obtained in different types of twins were compared with one another and also with the value obtained in single-born individuals.

(3) There are relatively more left-handed and left-footed individuals among twins than among single-born. However, there are not sufficient data for asserting that left-handedness and left-footedness are more frequent among monozygotic than among dizygotic twins.

(4) The reversed asymmetry in handedness and footedness is a phenomenon related somehow to twinning; but this reversing agency is probably something other than mirror-imaging.

(5) The relative frequency of the dextral, sinistral and double whorls in Japanese children is about (50+) : 40 : (10—). This value differs rather considerably from that known in white children where the frequency of the sinistral whorl is estimated as 18-20 per cent.

(6) There is no conclusive evidence that the sinistral whorl is commoner among twins than among single-born individuals, nor is there any reliable difference between different types of twins. Thus, no relation is found between twinning and the reversed direction of the head whorl.

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A STUDY ON THE FREQUENCY OF LEFT-HANDEDNESS AND LEFT-FOOTEDNESS AMONG JAPANESE SCHOOL CHILDREN

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INTRODUCTION

IN the course of our researches on the handedness of twins, it became necessary for us to find the frequency of left-handedness among children of the primary school ages. But no adequate data were available from published works. Further, it seemed desirable to obtain data with reference to the same criteria that we had employed for the study of the handedness of twins. This induced us to undertake the present inquiry. The results here presented, though rather inconclusive, may not be without value for those who are interested in the problem of handedness, especially as we have only fragmentary knowledge of the handedness of Japanese people.

The method we used in the present study was to give each child the following questions printed in the most intelligible form, and make him (her) write down the answer "right" or "left" for each item:

1. "Which hand do you use for striking a match?"
2. "In which hand do you hold your penknife in sharpening your pencil?"
3. "In which hand do you hold your scissors?"
4. "Which hand do you throw a ball with?"
5. "In which hand do you hold your chop-sticks?"
6. "In which hand do you hold your writing brush?"
7. "Which leg do you kick a foot-ball with?"

The answers were obtained from about 17,000 children of twenty primary schools in the city of Kyoto. The school-years ranged over the whole eight years of the primary school course from the first (lowest) year of the ordinary primary school to the second (highest) year of the higher primary school. The ages of the first year children of the

ordinary primary school at the time of this study were between 6 years 7 months and 7 years 6 months with only a few negligible exceptions, and those of the children of the higher grades were older by 1-7 years according to the grade of the classes to which the children belonged. In selecting the criteria for handedness and footedness, stated above, we followed the advice of Mr. T. Obonai, Professor of Psychology of the Tokyo Higher School, and in pursuing the study, we were helped considerably by the coöperation of the teachers of the primary schools.

The results obtained are presented in Tables 1-3 and Figures 1-2. Those who gave answer as "both right and left" are included in the left groups. These tables and graphs show that the frequency of left-handedness or footedness varies according to (1) criterion for detecting handedness or footedness, (2) age, and (3) sex of the children.

TABLE 1

Frequency of Left-handedness in Primary School Children as Determined With Reference to All Criteria

SCHOOL YEAR	SEX	NUMBER	LEFT-HANDED		SEXUAL INDEX
			Number	%	
I	♂	889	152	17.1 ± 1.26	100
	♀	1072	167	15.6 ± 1.11	91
II	♂	1345	189	14.1 ± 0.95	100
	♀	1256	158	12.6 ± 0.94	89
III	♂	1390	169	12.2 ± 0.88	100
	♀	1249	144	11.5 ± 0.90	94
IV	♂	1473	154	10.5 ± 0.80	100
	♀	1336	119	8.9 ± 0.78	85
V	♂	1350	146	10.8 ± 0.84	100
	♀	1161	91	7.8 ± 0.79	72
VI	♂	1231	160	13.0 ± 0.96	100
	♀	1151	111	9.6 ± 0.86	74
VII	♂	905	103	11.4 ± 1.06	100
	♀	403	33	8.2 ± 1.37	65
VIII	♂	489	46	9.4 ± 1.32	100
	♀	247	14	5.7 ± 1.48	61
Total	♂	9072	1119	12.3 ± 0.34	100
	♀	7875	837	10.6 ± 0.35	86

DIFFERENCE ACCORDING TO CRITERION

In Table 1 and Figure 1 all those children who described themselves as using their left hands for any one of the works mentioned above, were listed as left-handed. The frequency of left-handedness is as high as 17 per cent (♂) and 15.6 per cent (♀) in the youngest class; it becomes gradually lower in the older classes, in the oldest class being 9.4 per cent (♂), 5.7 per cent (♀). But if, on the other hand, the data for each criterion are taken separately (Table 2 and Figure 2), still lower values are obtained, which vary according to the criteria. Of the six criteria mentioned above, pencil-sharpening with a penknife and ball-throwing, which evidently require some skill and strength, show the

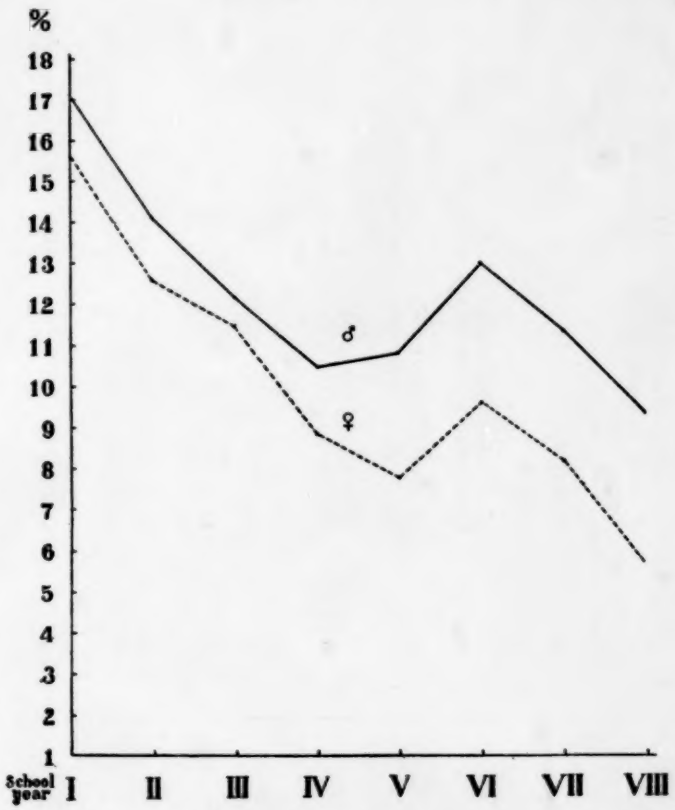

FIG. 1. GRAPH BASED ON THE DATA IN TABLE 1

TABLE 2

Frequency of Left-handedness and Left-footedness in Primary School Children as Determined With Reference to Each Criterion

SCHOOL YEAR	SEX	PENKNIFE	BALL	SCISSORS	MATCH	CHOPSTICKS	WRITING-BRUSH	FOOTBALL
I	♂	10.0 ± 1.08	7.4 ± 0.93	7.2 ± 0.92	5.9 ± 0.84	4.6 ± 0.75	5.1 ± 0.78	8.3 ± 0.99
	♀	7.4 ± 0.81	6.4 ± 0.75	4.4 ± 0.63	3.6 ± 0.59	1.9 ± 0.42	2.9 ± 0.51	5.6 ± 0.71
II	♂	6.0 ± 0.65	6.2 ± 0.66	3.6 ± 0.51	3.2 ± 0.48	2.2 ± 0.40	1.3 ± 0.31	6.1 ± 0.65
	♀	6.5 ± 0.71	4.6 ± 0.60	3.4 ± 0.51	3.2 ± 0.51	2.4 ± 0.43	1.8 ± 0.37	5.6 ± 0.66
III	♂	6.8 ± 0.68	6.5 ± 0.66	4.6 ± 0.56	3.4 ± 0.45	1.4 ± 0.32	0.6 ± 0.66	5.6 ± 0.62
	♀	4.0 ± 0.55	3.6 ± 0.54	3.0 ± 0.48	2.5 ± 0.44	2.1 ± 0.41	1.2 ± 0.31	5.5 ± 0.65
IV	♂	8.1 ± 0.71	8.5 ± 0.73	7.3 ± 0.68	6.5 ± 0.64	2.8 ± 0.43	1.0 ± 0.26	7.8 ± 0.70
	♀	4.4 ± 0.56	4.7 ± 0.58	3.6 ± 0.51	3.1 ± 0.47	1.8 ± 0.36	1.1 ± 0.29	4.0 ± 0.53
V	♂	5.5 ± 0.62	6.4 ± 0.67	5.3 ± 0.61	4.7 ± 0.58	1.8 ± 0.36	0.8 ± 0.24	5.9 ± 0.64
	♀	4.8 ± 0.62	4.3 ± 0.60	2.4 ± 0.45	3.0 ± 0.50	1.1 ± 0.31	0.9 ± 0.28	4.2 ± 0.59
VI	♂	6.4 ± 0.70	8.1 ± 0.78	6.4 ± 0.70	6.1 ± 0.68	2.0 ± 0.40	0.3 ± 0.16	7.1 ± 0.73
	♀	5.9 ± 0.69	5.2 ± 0.65	3.9 ± 0.57	3.5 ± 0.54	0.9 ± 0.28	0.3 ± 0.16	5.4 ± 0.67
VII	♂	6.5 ± 0.81	8.1 ± 0.91	6.3 ± 0.81	6.2 ± 0.80	1.4 ± 0.50	0.7 ± 0.33	6.5 ± 0.82
	♀	4.0 ± 0.98	5.0 ± 1.09	3.0 ± 0.85	3.0 ± 0.85	1.2 ± 0.54	0.2 ± 0.22	4.0 ± 0.97
VIII	♂	5.1 ± 0.99	6.4 ± 1.11	5.7 ± 1.05	4.3 ± 0.92	0.4 ± 0.29	0.2 ± 0.20	5.1 ± 1.00
	♀	4.9 ± 1.40	4.0 ± 1.25	4.5 ± 1.32	2.0 ± 0.89	0.8 ± 0.57	0.0 ± 0.00	3.2 ± 1.12

highest frequency (5-8.5 per cent ♂, 4-6 per cent ♀). Next come criteria like handling scissors and striking a match, for which less skill and strength are needed (3-7 per cent ♂, 2.5-3 per cent ♀). The frequency is lowest for chop-sticks and writing brush, which are much under parents' and teachers' training influence (0-3 per cent for both sexes). The frequency for footedness is nearly the same as for those activities mentioned first (5.5-8.5 per cent ♂, 3.5-6 per cent ♀).

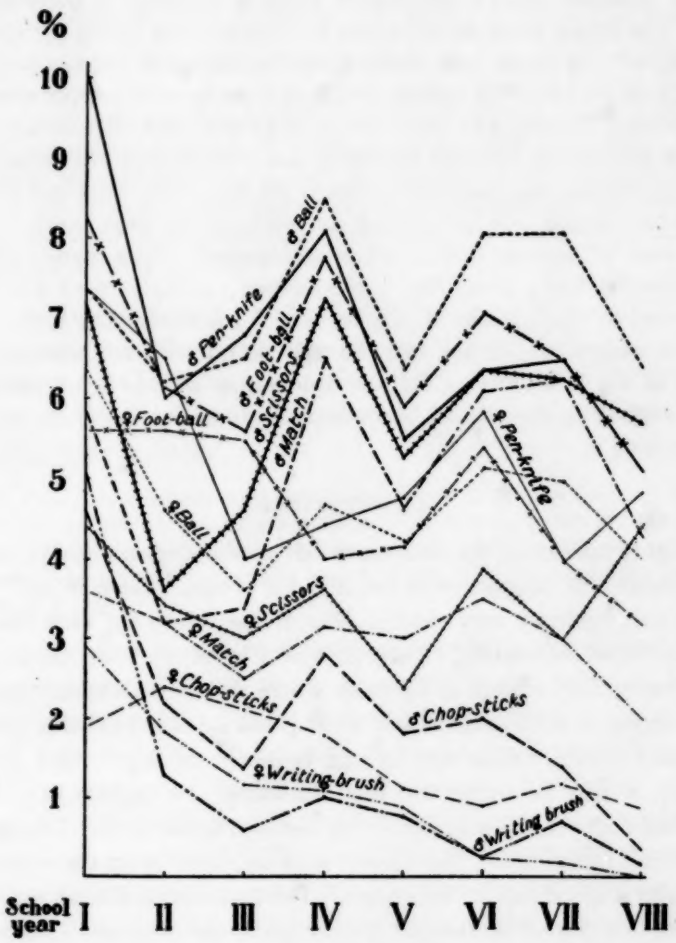

FIG. 2. GRAPH BASED ON THE DATA IN TABLE 2

The frequency of left-handedness as well as left-footedness thus varies considerably according to different criteria. Evidently this is mainly responsible for the great differences found among previous authors' estimates (see Siemens, 1925, and Ludwig, 1932). If by left-handedness is meant that the child has a natural inclination to prefer the left hand to the right for doing works which require some skill and strength, then the values estimated by such criteria as penknife or ball seem to be the best. The findings of previous authors, especially Stier (1911), Neurath (1922), and Kamm (1930), are much in favor of this view. The values obtained according to these criteria (5-8.5 per cent ♂, 4-6 per cent ♀) come near those given by Schaefer (1911; 5.15 per cent ♂, 2.98 per cent ♀), Parson (1924; 5.5 per cent ♂, 3.0 per cent ♀), and Neurath (1922; 4.43 per cent ♂, 3.42 per cent ♀), obtained by methods and among material similar to this case, though the former are somewhat higher than the latter. But if all those who show some traits of left-handedness are considered as left-handed, then much higher values such as those in Table 1 are to be expected. As a matter of fact, of previous authors, some like v. Bardeleben (at least 10-12 per cent) and Siemens (13.4 per cent ♂, 9.9 per cent ♀) give similarly high values for their materials. At any rate, there is no question but that the estimation of the frequency of left-handedness, as well as left-footedness, varies much with the idea of handedness or footedness, and the method for detecting it.

AGE DIFFERENCE

Roughly speaking, the frequency of left-handedness in the child's age diminishes in proportion to the advance of age. Table 1 and Figure 1 show this tendency very clearly. But if the values for each criterion are considered separately (Table 2 and Figure 2), this tendency is hardly recognized, except in the cases where the environmental influence is very strong, i. e. chop-sticks and writing-brush. For the other criteria, fluctuation of the values age by age is more striking. As a general tendency, it may be recognized that the values are highest in the first year, then comes an abrupt fall in the second, a rise in the fourth, and another fall in the fifth; then there comes another rise in the sixth year and finally a third fall in the eighth. The footedness also shows much the same fluctuating tendency. In the girls, the fluctuation is not so marked as in the boys; but the general tendency is identical, especially

the rise in the sixth year is rather remarkable. This rise is clearly recognizable even in Table 1 and Figure 1, where otherwise the decline is very gradual and regular.

The great height of the frequency in the first year suggests a still greater height in the pre-school ages such as shown by Bethe (1925). For the abrupt fall in the second year the teachers' influence is no doubt responsible. It is less easy to interpret the fluctuation in the following years. But evidently this is related to the fluctuation in the rate of the physical or psychical development of the child.

Such a fluctuation in the frequency of left-handedness is not mentioned in the works of previous authors, so far as we know, except that of the Japanese psychologist Tomita (1915), who investigated the left-handedness of school children by means of the relative efficiency of two hands in manipulating an abacus. This author found that the curve of frequency of left-handedness shows a remarkable rise at the age of eleven, and he interpreted this in connection with the recognized fact that there comes at about this age a period of retardation of development both physically and mentally.

The rise at the sixth year perceived in our material is probably a phenomenon with a similar significance.

As for the gradual decrease with age, such as found in Table 1 and Figure 1, two kinds of factors, internal and external, may be considered as working hand in hand.

Bethe (1925) has demonstrated clearly that before the age of four, the relative dexterity of one hand is yet indistinct; it develops largely in the two succeeding years, but a fraction of the children remain either ambidextrous or only very slightly one-handed; with the gradual growth of the child, the handedness becomes more firmly fixed.

This is apparently normal developmental change, which is mainly dependent on internal factors. But at the same time, environmental factors such as parents' and teachers' interference, the influence of tools and customs, come to work and intensify or hinder the development of the handedness determined by the internal factors. The rather regular decrease up to the fifth year in Table 1 and Figure 1, contrary to the considerable fluctuation in Table 2 and Figure 2, is largely due to the fact that in the younger classes there are more neutral individuals who use their left hands for only a few kinds of the works in question, and these raise the number of left-handed individuals; with the advance of

age, however, the handedness of each individual is more clearly fixed, and most of the children become either definite right- or definite left-handers, only a few remaining as neutral members.

SEXUAL DIFFERENCE

For almost all the criteria and ages, the frequency of left-handedness is greater in boys than in girls. As shown in Table 3, the sexual indices fall between 0.5 and 1, with a few exceptions. The footedness shows much the same tendency.

TABLE 3

Sexual Indices of the Frequency of Left-handedness and Left-footedness Based on the Data in Table 2, the Values in Boys Being Taken as 100

SCHOOL YEAR	PEN-KNIFE	BALL	SCISSORS	MATCH	CHOP-STICKS	WRITING-BRUSH	FOOT-BALL
I	75	87	60	63	42	57	66
II	107	73	94	93	109	139	89
III	59	55	65	77	150	200	96
IV	54	55	49	48	64	110	51
V	87	67	45	64	61	113	70
VI	92	64	61	58	45	100	76
VII	62	58	48	48	71	29	57
VIII	96	64	79	47	200	..	63
Average	79	65	63	62	93	107	71

The sexual difference in the frequency of left-handedness is universally recognized. But the estimate varies according to investigators. There are some, for instance, v. Bardeleben (1911), who deny this difference. Others (cf. Siemens, 1924) even maintain that left-handedness is commoner in females than in males. But more recent authors accord in the view that the frequency is greater in males than in females. The sexual index is usually put at about 0.5, but may be somewhat higher.

As shown in Table 3, in the matter of sexual difference also, chopsticks and writing-brush stand in contrast to other criteria. The sexual index is generally high in these two criteria; especially in the writing-

brush, where the average value of the sexual indices even exceeds 1.00. This fact implies that, although left-handedness of the ordinary grade is commoner in boys than in girls, the frequency of extreme left-handedness is about the same in the two sexes.

In Table 2 and Figure 2, it is shown that the sexual index is lower in the younger classes than in the older. This apparently favors the view held by some previous authors like v. Bardeleben that the born trait of left-handedness may be found among females as frequently as among males.

SUMMARY

1. The handedness and footedness of about 17,000 Japanese school children were determined by the questionnaire method in reference to seven criteria, and the frequency of left-handedness and footedness was estimated.

2. The frequency varies according to (1) criterion for handedness and footedness, (2) age, and (3) sex of the children.

3. The frequency is highest (♂ 5-8.5 per cent, ♀ 4-6 per cent) for the works which require some skill and strength, such as ball-throwing and pencil-sharpening, and lowest (0-3.0 per cent for both sexes) for the works which receive environmental interference most, such as chop-stick handling and writing with a brush. The frequency of left-footedness as determined by foot-ball-kicking, is nearly the same as for those hand-works mentioned first (♂ 5.5-8.5 per cent, ♀ 3.5-6 per cent).

4. The frequency of the individuals who show the trait of left-handedness decreases gradually with age except for a rise at the sixth school year. But if the handedness is determined with reference to a single criterion, the frequency does not show such a regular decrease and the fluctuation age by age is more marked. An especially striking rise occurs at the sixth school year for every criterion.

5. The frequency of left-handedness is 1-2 times as high in boys as in girls; for extreme left-handedness, however, the frequency is about the same in the two sexes.

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THE HISTORY OF LONGEVITY IN THE UNITED STATES*

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I

WHAT has been the gain in the expectation of life in the United States in the past fifty, or the past one hundred years? Has this gain been due wholly to the saving of infants and young people from death, or has there been gain over the entire stretch of life? Or, has there by any chance, been actual loss at certain ages? And to what causes shall we attribute such gains or losses as have taken place? These are questions that have been agitated in recent times not only by specialists who devote their efforts to the study of vital statistics, but also by the general public, as we see very clearly from newspaper notices and from inquiries that come to us in our insurance company almost daily.

The recently published details of the findings of the United States Census of 1930 add a particular interest to these questions at the present time, for it is only in a census year that direct observations are available from which the answers to the questions propounded above can be ascertained with some precision. The forthcoming publication of a volume by the authors dealing with life tables on the basis of materials collected at the recent and preceding censuses, furnishes the occasion for a retrospective study. By contrasting the new with the earlier life tables, we shall be led to view the development of longevity among our people in the past, and by performing certain special computations, we shall be given clues regarding the part taken by various causes of death in the curtailment of human life. Finally, we may find that a comparison with conditions observed abroad helps to throw light on our inquiries.

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* Paper read on December 27, 1932 at the Atlantic City meeting of the American Association for the Advancement of Science, Section K (Social and Economic Sciences).

In tracing the history of longevity in the United States we find ourselves handicapped by a great scarcity of data in the early periods. Our retrospect here must necessarily be sketchy and we must seek to make the best of such fragments of information as are available. Actual systematic records of current mortality for any considerable area in the United States are a matter of very recent origin. The Bureau of the Census, which today compiles and publishes statistics of mortality, dates, as a permanent institution, practically from the census of 1900, having previously functioned sporadically only at the time of actual census-taking. While we have fairly accurate censuses beginning with 1790, the facts of mortality have not been currently available until the beginning of the present century.

Among the earliest attempts to prepare life tables for the populations of American communities is a series of survival tables communicated by William Barton to the American Philosophical Society on March 18, 1791. These cover a number of cities, among them two in the United States; namely, Philadelphia, Pa. 1782-1790, and Salem, Mass. 1782-1790. These two American survival tables are essentially of the nature of historical curiosities. Their documentary value is slight. For our purpose it is sufficient to say that the expectation of life at birth in Philadelphia according to Barton's observations seems to have been about 25 years. At about the same time (1789) there appeared the Wigglesworth Tables giving an expectation of life for Massachusetts and New Hampshire of about 35 years. For the year 1814 two life tables were prepared by the Pennsylvania Company for Insurance upon Lives, one based on the records of the Philadelphia board of health, the other on records of the Episcopal Church. The former gives the expectation of life at age one as 25.96, the latter as 30.91. The reliability of these tables also is questionable. We may surmise from this evidence and from comparative figures for European centers living under similar sanitary conditions that for the country as a whole at the end of the eighteenth century the expectation of life at birth must have been about 30 years.

The very incomplete picture given us in these early life tables is pieced out, in a way, by more or less fragmentary and inaccurate data which we have of deathrates prevailing in certain cities in the early part of the last century. For example, old records for New York City indicate a deathrate of 28.7 for the year 1804, followed by a series of rates which for the most part move between 20 and 30 and nearer to 30

TABLE 1

Deathrate from All Causes per 1,000 Population, New York, N. Y. and Philadelphia, Pa.

YEAR	NEW YORK	YEAR	PHILADELPHIA
1804	28.72	....	....
1810	21.60	....	....
1815	25.12	....	....
1820	28.57	....	....
1825	29.62	....	....
1830	27.13	....	....
1835	26.27	....	....
1840	27.01	1840	52.84
1845	27.27	1845	58.05
1850	30.60	1850	70.10

up to 1850 and even later. A series of such rates is shown in Table 1 and the accompanying graph, Figure 1. The early deathrates were often exceptionally high in years when there happened to be an epidemic of cholera, yellow fever or other catastrophic outbreak of disease. In such years the deathrate rose to such excessive heights as 45, 46 and even 47

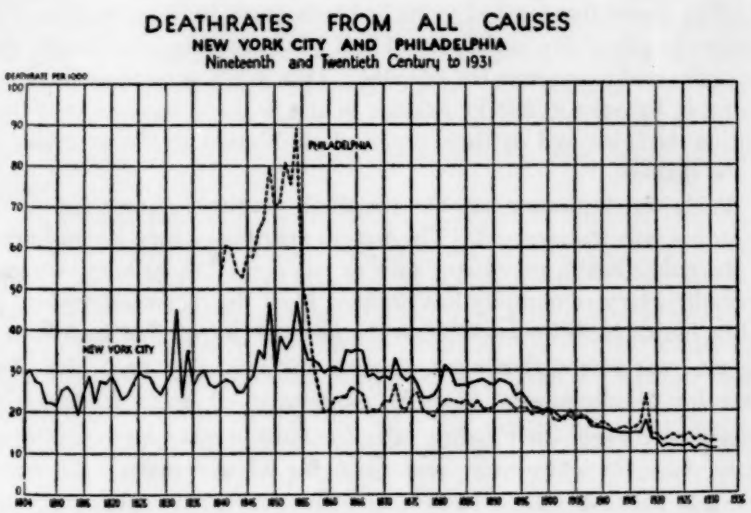


FIG. 1

per 1,000, as indicated in the records, which themselves undoubtedly understate the actual conditions, for registration of deaths in these early days must have been decidedly inadequate. It is only after the middle of the last century that definitely better deathrates began to show themselves.

From about the middle of the nineteenth century American life tables become somewhat abundant.

II

The State of Massachusetts has a more extended series of life tables than any other area in the United States. A life table for this State, based on very imperfect data for the year 1850, was constructed by Joseph Kennedy, who had been Superintendent of the United States Census in 1850. This gave an expectation of life at birth of 38.3 years for males and 40.5 for females. Then followed the Elliott life table for 1855 which gave an expectation of life at birth of 39.8 for persons of both sexes combined; the Billings table (1878-1882) with an expectation of life of 41.74 for males and 43.50 for females; the Glover table (1890), with 42.50 for males and 44.46 for females; the Abbott table (1893-1897) with 44.09 for males and 46.61 for females; the Glover life table for 1901 with 46.07 for males and 49.42 for females. This series of tables shows that marked gains had been made in the extension of the average length of life in the second half of the century, about eight years for males and nine years for females. This is not surprising in view of the rapid increase in our knowledge in the fields of sanitation and preventive medicine and of their practical application to the everyday life of the masses.

With the beginning of the twentieth century, a new era in life extension was inaugurated. The last 30 years have seen a development of the public health movement such as has never before been witnessed. Virtually every community has profited from the increased medical and public health facilities available to the people. So we find each succeeding ten year period showing a sizeable advance. By 1910, the Glover table for Massachusetts shows an expectation of 49.33 for males and 53.06 for females; the Foudray table for 1919 to 1920, an expectation of 54.07 years for white males and 56.56 for white females. To this we now add the table computed by us for the year 1930, which gives 59.48 years for males and 62.74 years for females. In the last 30 years there

has been a gain in the average length of life of about thirteen and one-half years for each of the two sexes. Such an achievement was never before recorded.

Let us consider the series of Massachusetts life tables from 1890 to 1930 a little more in detail. Concentrating our attention first of all on the column headed age zero in Table 2, we note that the gains in the expectation of life have with one or two unimportant exceptions been greater among females than among males for this forty year period and at all ages shown in the table.

Secondly, we observe that the gains from decade to decade decrease rapidly with advancing age until at age forty they virtually disappear altogether. After age forty there are even a few minus signs within this forty year period, signifying slight decreases in the expectation of life. But none of these occur within the last three decades, that is, in the range of the year 1930 compared with 1920, 1910 or 1901. In these calendar periods there is an increase shown for every item. Where decreases appear in other parts of the table, they are with few exceptions smaller in amount among females than among males.

More direct indication of the gains made at each age x is given by q_x, the mortality at age x (or to be more exact the probability at age x of dying within one year) than by e_x, the expectation of life, because this latter is of the nature of an average taken over the whole range from age x to the end of life, whereas q_x measures conditions *at* each particular age. Accordingly, Table 3 has been prepared, in which are collected together the main facts regarding q_x for the State of Massachusetts according to those life tables for which data are available in sufficient detail. This means that in this comparison we must begin at the year 1890.

A glance over Table 3 brings out at once the familiar fact that the mortality is extremely high in the first year of life (q_0), rapidly falls to a low minimum at or about the age of ten, then rises at first slowly, and then, as the sixtieth year of age is reached, ascends quite rapidly to reach again high values at age eighty and beyond. While the actual figures differ greatly in successive decennia, this general picture holds in every case.

Concentrating our attention again on the first year of life, as characterized by figures for q_0 appearing in the column headed age zero of the table, we note that between the two extremes of the calendar period tabulated, the figure for males has dropped almost to a third of

TABLE 2

Expectation of Life Among Males and Females in Massachusetts in 1850, 1890, 1901, 1910, 1920, 1930 Together with Gain or Loss in Expectation Over Certain Periods—For the Decennial Ages of Life

SEX AND CALENDAR PERIOD	AGE								
	0	10	20	30	40	50	60	70	80
	Complete Expectation of Life (e_x) at Age x.								
Males—									
1850	38.3	48.0	40.1	34.0	27.9	21.6	15.6	10.2	5.9
1890	42.50	48.45	40.66	34.05	27.37	20.72	14.73	9.35	5.40
1901	46.07	50.17	41.82	34.46	27.17	20.15	13.90	8.88	5.07
1910	49.33	51.14	42.48	34.55	26.97	19.79	13.42	8.58	5.07
1919-20*	54.07	53.27	44.58	36.57	28.78	21.19	14.35	8.89	5.02
1930	59.48	55.37	46.24	37.51	29.13	21.26	14.49	9.11	5.31
Females—									
1850	40.5	47.2	40.2	35.4	29.8	23.5	17.0	11.3	6.4
1890	44.46	49.62	42.03	35.36	28.76	22.09	15.70	10.15	5.75
1901	49.42	52.12	43.71	36.19	28.79	21.57	15.06	9.56	5.55
1910	53.06	53.56	44.85	36.78	29.04	21.55	14.79	9.49	5.49
1919-20*	56.56	54.32	45.53	37.75	30.03	22.32	15.28	9.56	5.27
1930	62.74	57.93	48.66	39.89	31.32	23.13	15.84	9.93	5.72

* White population only.

TABLE 2—*Continued*

SEX AND CALENDAR PERIOD	AGE								
	0	10	20	30	40	50	60	70	80
	Increase (+) or Decrease (—) in Expectation of Life								
Males—									
1901 over 1890	+ 3.57	+ 1.72	+ 1.16	+ .41	— .20	— .57	— .83	— .47	— .33
1910 over 1890	+ 6.83	+ 2.69	+ 1.82	+ .50	— .40	— .93	— 1.31	— .77	— .33
1919-20 over 1890	+11.57	+ 4.82	+ 3.92	+ 2.52	+ 1.41	+ .47	— .38	— .46	— .38
1930 over 1850	+21.2	+ 7.4	+ 6.1	+ 3.5	+ 1.2	— .3	— 1.1	— 1.1	— .6
1930 over 1890	+16.98	+ 6.92	+ 5.58	+ 3.46	+ 1.76	+ .54	— .24	— .24	— .09
1930 over 1901	+13.41	+ 5.20	+ 4.42	+ 3.05	+ 1.96	+ 1.11	+ .59	+ .23	+ .24
1930 over 1910	+10.15	+ 4.23	+ 3.76	+ 2.96	+ 2.16	+ 1.47	+ 1.07	+ .53	+ .24
1930 over 1919-20	+ 5.41	+ 2.10	+ 1.66	+ .94	+ .35	+ .07	+ .14	+ .22	+ .29
Females—									
1901 over 1890	+ 4.96	+ 2.50	+ 1.68	+ .83	+ .03	— .52	— .64	— .59	— .20
1910 over 1890	+ 8.60	+ 3.94	+ 2.82	+ 1.42	+ .28	— .54	— .91	— .66	— .26
1919-20 over 1890	+12.10	+ 4.70	+ 3.50	+ 2.39	+ 1.27	+ .23	— .42	— .59	— .48
1930 over 1850	+22.2	+10.7	+ 8.5	+ 4.5	+ 1.5	— .4	— 1.2	— 1.4	— .7
1930 over 1890	+18.28	+ 8.31	+ 6.63	+ 4.53	+ 2.56	+ 1.04	— .14	— .22	— .03
1930 over 1901	+13.32	+ 5.81	+ 4.95	+ 3.70	+ 2.53	+ 1.56	+ .78	+ .37	+ .17
1930 over 1910	+ 9.68	+ 4.37	+ 3.81	+ 3.11	+ 2.28	+ 1.58	+ 1.05	+ .44	+ .23
1930 over 1919-20	+ 6.18	+ 3.61	+ 3.13	+ 2.14	+ 1.29	+ .81	+ .56	+ .37	+ .45

TABLE 3

Annual Rates of Mortality per Thousand Among Males and Females in Massachusetts in 1890, 1901, 1910, 1919-20, 1930, Together with Increase or Decrease in Mortality Rates over Certain Periods at Decennial Ages

SEX AND CALENDAR PERIOD	AGE								
	0	10	20	30	40	50	60	70	80
	Probability per Thousand ($1000q_x$), at Age x, of Dying Within One Year								
Males—									
1890	167.77	3.99	7.75	9.96	12.09	17.41	28.87	56.66	121.07
1901	158.78	2.74	5.84	7.80	10.60	16.11	32.16	62.59	134.81
1910	137.06	2.37	4.70	6.60	10.00	16.05	33.15	67.91	137.43
1919-20*	98.62	2.14	3.88	6.06	7.96	12.86	26.46	62.31	130.70
1930	63.14	1.42	2.59	3.67	6.57	12.54	27.67	58.54	132.53
Females—									
1890	147.55	4.01	7.47	9.68	11.72	15.06	25.88	50.20	108.97
1901	131.18	2.47	5.09	7.33	9.51	14.04	26.86	56.47	117.14
1910	113.04	2.11	3.89	5.97	8.14	12.58	27.03	56.47	123.49
1919-20*	78.26	1.79	4.11	6.44	7.19	11.87	23.19	54.28	119.67
1930	52.23	1.05	2.23	3.63	4.67	10.07	21.31	50.60	117.05

* White population only.

TABLE 3—*Continued*

SEX AND CALENDAR PERIOD	AGE								
	0	10	20	30	40	50	60	70	80
	Increase (+) or Decrease (—) in $1000q_x$								
Males—									
1901 over 1890	— 8.99	—1.25	—1.91	—2.16	—1.49	—1.30	+3.29	+ 5.93	+13.74
1910 over 1890	— 30.71	—1.62	—3.05	—3.36	—2.09	—1.36	+4.28	+11.25	+16.36
1919-20 over 1890	— 69.15	—1.85	—3.87	—3.90	—4.13	—4.55	—2.41	+ 5.65	+ 9.63
1930 over 1890	—104.63	—2.57	—5.16	—6.29	—5.52	—4.87	—1.20	+ 1.88	+11.46
1930 over 1901	— 95.64	—1.32	—3.25	—4.13	—4.03	—3.57	—4.49	— 4.05	— 2.28
1930 over 1910	— 73.92	— .95	—2.11	—2.93	—3.43	—3.51	—5.48	— 9.37	— 4.90
1930 over 1919-20	— 35.48	— .72	—1.29	—2.39	—1.39	— .32	+1.21	— 3.77	+ 1.83
Females—									
1901 over 1890	— 16.37	—1.54	—2.38	—2.35	—2.21	—1.02	+ .98	+ 6.27	+ 8.17
1910 over 1890	— 34.51	—1.90	—3.58	—3.71	—3.58	—2.48	+1.15	+ 6.27	+14.52
1919-20 over 1890	— 69.29	—2.22	—3.36	—3.24	—4.53	—3.19	—2.69	+ 4.08	+10.70
1930 over 1890	— 95.32	—2.96	—5.24	—6.05	—7.05	—4.99	—4.57	+ .40	+ 8.08
1930 over 1901	— 78.95	—1.42	—2.86	—3.70	—4.84	—3.97	—5.55	— 5.87	— .09
1930 over 1910	— 60.81	—1.06	—1.66	—2.34	—3.47	—2.51	—5.72	— 5.87	— 6.44
1930 over 1919-20	— 26.03	— .74	—1.88	—2.81	—2.52	—1.80	—1.88	— 3.68	— 2.62

its original value, namely, from 168 in 1890 to 63 in 1930, for males, and from 148 in 1890 to 52 in 1930 for females. Observing now, not ratios, but differences, we note that in the period 1890 to 1930 the chances of dying within the first year of life have decreased by 104.6 per 1,000 among males and 95.3 per 1,000 among females. These improvements have been distributed over successive calendar periods as follows: 1890 to 1901, 9.0 for males and 16.4 for females; 1901 to 1910, 21.7 for males and 18.1 for females; 1910 to 1920, 38.4 for males and 34.8 for females; 1920 to 1930, 35.5 for males and 26.0 for females.

In studying as a whole the lower panel of the table, which exhibits the improvements in various calendar periods, the first feature that immediately strikes the eye is that at all ages under fifty there has been an improvement in every calendar period shown. At age sixty we begin to observe some plus signs, which, when we are speaking of mortalities, represent retrogression. One of these plus signs appears at age sixty for males for the last decade 1919-1920 to 1930. The retrogression here, however, is quite small. There are also plus signs at age sixty for males and females for the first two decades of the period covered by the table. At ages seventy and eighty plus signs become more frequent but are restricted almost wholly to the comparisons with 1890.

Among females improvement is shown at every age and for all calendar periods since 1901 appearing in the table. Among males the situation is a trifle less favorable for, as already noted, retrogression appears at ages sixty and eighty in the last calendar period.

III

Table 4 and Figures 2 and 3 exhibit, for the ten Original Death Registration States as constituted in 1900, the data corresponding, so far as they are available, to those shown in Table 2 for Massachusetts. The series necessarily begins with the Glover life table for the year 1901. Again concentrating our attention first of all on the column headed age zero, i.e. on the expectation of life at birth, we note that in the period 1901 to 1930 there has been an increase from 48.23 years to 59.09 years for white males, that is, a gain of 10.86 years; for white females there is an increase from 51.08 years to 62.62 years, a gain of 11.54 years. These increases have been distributed over successive calendar periods as follows: 1901 to 1910, white males, 2.00 years, white females 2.54 years;

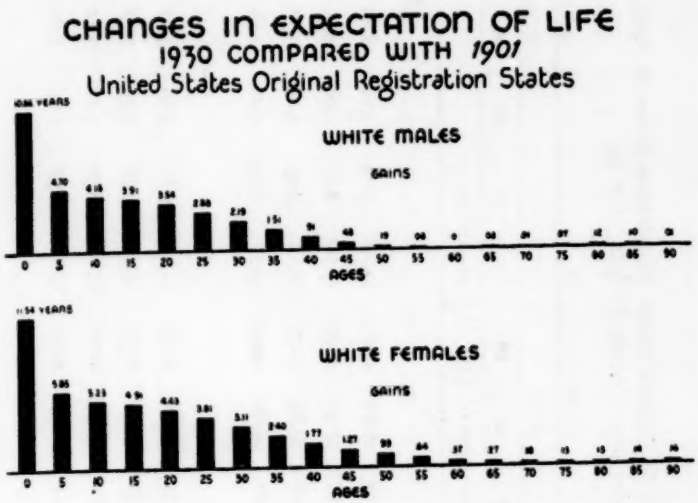

CHANGES IN EXPECTATION OF LIFE
1930 COMPARED WITH 1901
United States Original Registration States
WHITE MALES
GAINS
AGES
WHITE FEMALES
GAINS
AGES

FIG. 2

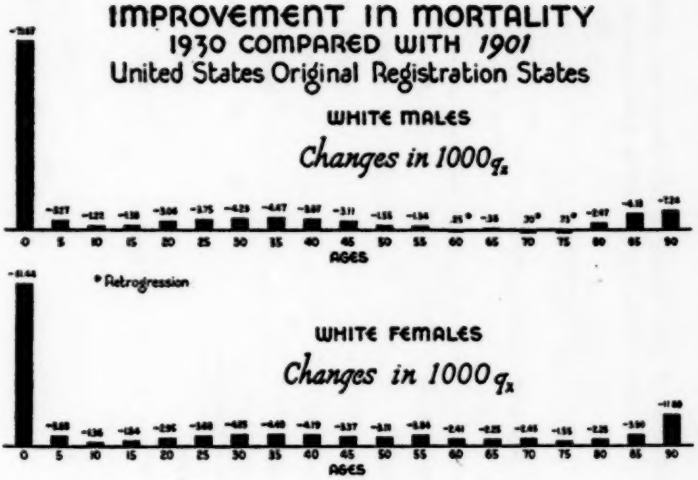

IMPROVEMENT IN MORTALITY
1930 COMPARED WITH 1901
United States Original Registration States
WHITE MALES
Changes in 1000 q_x
AGES
* Retrogression
WHITE FEMALES
Changes in 1000 q_x
AGES

FIG. 3

TABLE 4

Expectation of Life Among White Males and White Females in U. S. Original Death Registration States in 1901, 1910, 1920, 1930, Together with Gain or Loss in Expectation over Certain Periods. For the Decennial Ages of Life

SEX AND CALENDAR PERIOD	AGE									
	0	10	20	30	40	50	60	70	80	90
	Complete Expectation of Life ($\mathring{e}_x$) at Age x.									
White Males—										
1901	48.23	50.59	42.19	34.88	27.74	20.76	14.35	9.03	5.10	2.85
1910	50.23	51.32	42.71	34.87	27.43	20.39	13.98	8.83	5.09	2.99
1919-20	54.05	52.82	44.29	36.47	28.85	21.37	14.62	9.09	5.19	2.75
1930	59.09	54.75	45.73	37.07	28.65	20.95	14.35	9.04	5.22	2.86
White Females—										
1901	51.08	52.15	43.77	36.42	29.17	21.89	15.23	9.59	5.50	3.02
1910	53.62	53.57	44.88	36.96	29.26	21.74	14.92	9.38	5.35	3.00
1919-20	56.41	53.82	45.16	37.61	29.95	22.26	15.29	9.59	5.57	3.11
1930	62.62	57.38	48.20	39.53	30.94	22.82	15.60	9.77	5.63	3.18

TABLE 4—*Continued*

SEX AND CALENDAR PERIOD	AGE									
	0	10	20	30	40	50	60	70	80	90
	Increase (+) or Decrease (—) in Expectation of Life									
White Males—										
1910 over 1901	+ 2.00	+ .73	+ .52	— .01	— .31	— .37	— .37	— .20	— .01	+ .14
1919-20 over 1901	+ 5.82	+2.23	+2.10	+1.59	+1.11	+ .61	+ .27	+ .06	+ .09	— .10
1930 over 1901	+10.86	+4.16	+3.54	+2.19	+ .91	+ .19	+ .00	+ .01	+ .12	+ .01
1930 over 1910	+ 8.86	+3.43	+3.02	+2.20	+1.22	+ .56	+ .37	+ .21	+ .13	— .13
1930 over 1919-20	+ 5.04	+1.93	+1.44	+ .60	— .20	— .42	— .27	— .05	+ .03	+ .11
White Females—										
1910 over 1901	+ 2.54	+1.42	+1.11	+ .54	+ .09	— .15	— .31	— .21	— .15	— .02
1919-20 over 1901	+ 5.33	+1.67	+1.39	+1.19	+ .78	+ .37	+ .06	+ .00	+ .07	+ .09
1930 over 1901	+11.54	+5.23	+4.43	+3.11	+1.77	+ .93	+ .37	+ .18	+ .13	+ .16
1930 over 1910	+ 9.00	+3.81	+3.32	+2.57	+1.68	+1.08	+ .68	+ .39	+ .28	+ .18
1930 over 1919-20	+ 6.21	+3.56	+3.04	+1.92	+ .99	+ .56	+ .31	+ .18	+ .06	+ .07

TABLE 5

Annual Rates of Mortality per Thousand Among White Males and White Females in U. S. Original Death Registration States in 1901, 1910, 1920, 1930 Together with Increase or Decrease in Mortality Rates over Certain Periods at Decennial Ages

SEX AND CALENDAR PERIOD	AGE									
	0	10	20	30	40	50	60	70	80	90
	Probability per Thousand ($1000q_x$), at Age x, of Dying Within One Year									
White Males—										
1901	133.45	2.74	5.94	7.99	10.60	15.37	28.59	58.94	133.53	262.78
1910	123.26	2.38	4.89	6.60	10.22	15.53	30.75	62.14	135.75	255.17
1919-20	92.43	2.27	4.59	6.47	8.44	13.02	26.48	59.00	129.63	253.60
1930	61.78	1.52	2.88	3.76	6.73	13.82	28.84	59.64	131.06	255.54
White Females—										
1901	110.61	2.46	5.54	7.72	9.31	13.37	25.06	53.69	121.15	245.32
1910	102.26	2.06	4.20	6.03	8.03	12.59	25.83	56.63	125.79	247.59
1919-20	73.61	1.91	4.64	6.84	7.32	11.77	23.64	54.74	119.88	240.40
1930	49.17	1.10	2.59	3.47	5.12	10.26	22.65	51.24	118.90	233.63

TABLE 5—*Continued*

SEX AND CALENDAR PERIOD	AGE									
	0	10	20	30	40	50	60	70	80	90
	Increase (+) or Decrease (—) in $1000q_x$									
White Males—										
1910 over 1901	—10.19	— .36	—1.05	—1.39	— .38	+ .16	+2.16	+3.20	+2.22	—7.61
1919-20 over 1901	—41.02	— .47	—1.35	—1.52	—2.16	—2.35	—2.11	+ .06	—3.90	—9.18
1930 over 1901	—71.67	—1.22	—3.06	—4.23	—3.87	—1.55	+ .25	+ .70	—2.47	—7.24
1930 over 1910	—61.48	— .86	—2.01	—2.84	—3.49	—1.71	—1.91	—2.50	—4.69	+ .37
1930 over 1919-20	—30.65	— .75	—1.71	—2.71	—1.71	+ .80	+2.36	+ .64	+1.43	+1.94
White Females—										
1910 over 1901	— 8.35	— .40	—1.34	—1.69	—1.28	— .78	+ .77	+2.94	+4.64	+ 2.27
1919-20 over 1901	—37.00	— .55	— .90	— .88	—1.99	—1.60	—1.42	+1.05	—1.27	— 4.92
1930 over 1901	—61.44	—1.36	—2.95	—4.25	—4.19	—3.11	—2.41	—2.45	—2.25	—11.69
1930 over 1910	—53.09	— .96	—1.61	—2.56	—2.91	—2.33	—3.18	—5.39	—6.89	—13.96
1930 over 1919-20	—24.44	— .81	—2.05	—3.37	—2.20	—1.51	— .99	—3.50	— .98	— 6.77

1910 to 1919-1920 white males 3.82 years, white females 2.79 years; 1919-1920 to 1930, white males 5.04 years, white females 6.21 years.

In studying the table as a whole, that is, paying attention to the successive decennial ages, we note again that, in general, the gains in expectation of life have been greater among females than among males. There is, however, one systematic exception. In the twenty year period 1901 to 1919-1920 the gains are, at all ages shown, greater among males than among females. This table, like that for Massachusetts, exhibits some minus signs, indicating a retrogression in the expectation of life. Among females, however, these are restricted to the first calendar period, 1901 to 1910, ages 50 and over. Among males they occur in the same calendar period, ages 30 and over, and in the last calendar period, 1919-1920 to 1930, ages 40, 50, 60 and 70.

As was pointed out in discussing the series of life tables for the State of Massachusetts, more direct indication of the gains made at each age x of life is given by q_x, the mortality at age x, than by e_x, the expectation of life at the same age. Accordingly, as in the case of Table 3 for Massachusetts, there has been constructed also a summary Table 5 showing the values of q_x and the changes in q_x at decennial ages of life and in certain calendar periods. The range of calendar years covered is in this case from 1901 to 1930.

The general character of the facts brought out by this table is naturally very similar to that which was observed in the corresponding table for Massachusetts. At age zero in the period from 1901 to 1930 the mortality among white males has fallen to less than half its original value, the figures being 133.5 per 1,000 in 1901 and 61.8 per 1,000 for 1930, which corresponds to an improvement of 71.7 per 1,000. For females the figures are 110.6 in 1901 and 49.2 in 1930, representing an improvement of 61.4 per 1,000. These changes are distributed over successive decennia in the following fashion: in the decennium 1901 to 1910 an improvement of 10.2 for males and 8.4 for females; in the decennium 1910 to 1919-1920 an improvement of 30.8 for males and 28.7 for females; in the decennium 1919-1920 to 1930 an improvement of 30.7 for males and 24.4 for females.

In studying the lower panel of the table, which exhibits the improvement in various calendar years, it is again seen, as in the case of Massachusetts, that below age fifty there has been an improvement at every age and every calendar period shown. In the case of females the improvement extends up to age fifty at all calendar periods shown, and,

in fact, plus signs, indicating retrogression, are rare occurrences in that part of the table which relates to females; they are found only in the first decade and a solitary instance in the second decade at age seventy. Among males retrogression is shown in rather more numerous instances in various parts of the table above age fifty.

A comparison of the expectation of life between the years 1930 and 1920 gives an incomplete picture of the dominant situation, for a reason which becomes apparent on inspection of the graph, Figure 4. This

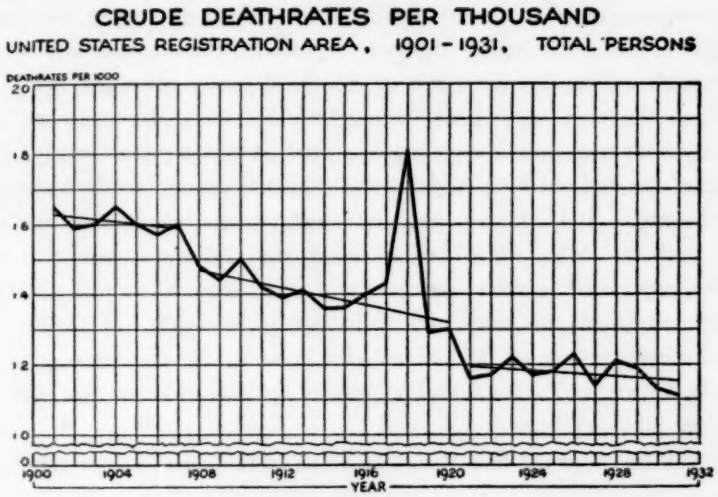


FIG. 4

graph exhibits a series of deathrates for the United States Death Registration Area over the period 1901 to 1931. This entire period obviously breaks up into three sub-periods, namely, 1901 to 1907 inclusive, 1908 to 1920 inclusive (but exclusive of the entirely atypical influenza year 1918); and lastly, the period 1921 to 1931 inclusive. Each of the three groups of years enumerated is characterized by a relatively moderate downward trend and by a separate and distinct average level, as follows:

Calendar Period	Deathrates	
	Average Level	Mean Annual Decrement
1901 to 1907	16.1	—.068 ± .055
1908 to 1920 (exclusive of 1918)	13.9	—.136 ± .021
1921 to 1931	11.7	—.042 ± .035

Of the three slopes only the second one is significant. The point of particular interest here is that the year 1920 happens to be the end of one of the three groups of years into which the series naturally splits up, while 1921 is the beginning of the next group. Between them is a rather sudden drop, very considerably greater than the usual fluctuation between two adjoining years. The consequence of this is that if we compare the expectation of life in the year 1930 with that in the year 1920, we get a markedly different result from that which is obtained by a comparison of 1930 with 1921. This is entirely proper, because in one case we are comparing the year 1930 with the earliest year of the series with a level characteristic of the third group of years, the group to which the year 1930 itself belongs; while when we compare 1930 with 1920 we compare it with the last year of the previous group, characterized throughout by a higher level than the group to which the year 1930 belongs. It is entirely proper to make both comparisons and keep them both in sight, remembering what the difference between them implies, especially as the years 1919 and 1920 were affected by a minor recrudescence of the epidemic of influenza of 1918. Accordingly a separate comparison of the values of e_x and q_x for the year 1930 and 1921 is shown in Tables 6 and 7, where, for convenience, the data for 1920 are also exhibited.

It will be observed that the changes in both q_x and e_x are much less extreme when comparison is made between 1930 and 1921 than when it is made between 1930 and 1920. At age zero the gain in expectation of life for white males appears as 5.0 years in the comparison 1919-1920 with 1930, but only as 2.4 years in the comparison 1921 with 1930. Similarly, in the case of females, the first comparison gives a gain of 6.2 years, the second only 3.5 years. From age ten on, throughout all ages exhibited, white males show actual losses in expectation of life, when the comparison is made between 1921 and 1930, whereas the comparison between 1919-1920 and 1930 shows minor losses only at ages forty to seventy. In the case of females the gains are all reduced, some of them to small negative values, when the basis of comparison with 1930 is changed from 1919-1920 to 1921. Similar observations are made on inspection of Table 7, which exhibits the corresponding values of q_x and changes therein.

TABLE 6

Expectation of Life Among White Males and White Females in U. S. Original Death Registration States in 1919-20, 1921 and 1930, Together with Gain or Loss in Expectation Over This Period

SEX AND CALENDAR PERIOD	AGE									
	0	10	20	30	40	50	60	70	80	90
	Complete Expectation of Life ($\mathring{e}_x$) at Age x.									
White Males—										
1919-20	54.05	52.82	44.29	36.47	28.85	21.37	14.62	9.09	5.19	2.75
1921	56.74	55.08	46.42	38.02	29.75	21.95	15.05	9.44	5.42	3.11
1930	59.09	54.75	45.73	37.07	28.65	20.95	14.35	9.04	5.22	2.86
White Females—										
1919-20	56.41	53.82	45.16	37.61	29.95	22.26	15.2	9.59	5.57	3.11
1921	59.11	56.07	47.28	38.96	30.72	22.80	15.68	9.90	5.72	3.20
1930	62.62	57.38	48.20	39.53	30.94	22.82	15.60	9.77	5.63	3.18
	Increase (+) or Decrease (—) in Expectation of Life									
White Males—										
1930 over 1919-20	+5.04	+1.93	+1.44	+ .60	— .20	— .42	— .27	— .05	+ .03	+ .11
1930 over 1921	+2.35	— .33	— .69	— .95	—1.10	—1.00	— .70	— .40	— .20	— .25
White Females—										
1930 over 1919-20	+6.21	+3.56	+3.04	+1.92	+ .99	+ .56	+ .31	+ .18	+ .06	+ .07
1930 over 1921	+3.51	+1.31	+ .92	+ .57	+ .22	+ .02	— .08	— .13	— .09	— .02

TABLE 7

Annual Rates of Mortality per 1,000 Among White Males and White Females in U. S. Original Death Registration States in 1919-20, 1921 and 1930, Together with Increase or Decrease in Mortality Rates Over This Period

SEX AND CALENDAR PERIOD	AGE									
	0	10	20	30	40	50	60	70	80	90
	Probability per Thousand ($1000q_x$), at Age x, of Dying Within One Year									
White Males—										
1919-20	92.43	2.27	4.59	6.47	8.44	13.02	26.48	59.00	129.63	253.60
1921	87.08	2.33	3.40	4.33	6.63	11.87	25.25	55.38	125.06	237.56
1930	61.78	1.52	2.88	3.76	6.73	13.82	28.84	59.64	131.06	255.54
White Females—										
1919-20	73.61	1.91	4.64	6.84	7.32	11.77	23.64	54.74	119.88	240.40
1921	69.25	1.90	3.33	4.48	6.06	10.76	22.85	51.10	115.88	229.42
1930	49.17	1.10	2.59	3.47	5.12	10.26	22.65	51.24	118.90	233.63
	Increase (+) or Decrease (—) in $1000q_x$									
White Males—										
1930 over 1919-20	—30.65	— .75	—1.71	—2.71	—1.71	+ .80	+2.36	+ .64	+1.43	+ 1.94
1930 over 1921	—25.30	— .81	— .52	— .57	+ .10	+1.95	+3.59	+4.26	+6.00	+17.98
White Females—										
1930 over 1919-20	—24.44	— .81	—2.05	—3.37	—2.20	—1.51	— .99	—3.50	— .98	— 6.77
1930 over 1921	—20.08	— .80	— .74	—1.01	— .94	— .50	— .20	+ .14	+3.02	+ 4.21

IV

QUALITATIVE CHANGES IN MORTALITY

Such are some of the facts that the bare life table figures tell. But our curiosity is not satisfied. We have further questions to ask. A deathrate is not completely defined by a single figure. It has qualitative characteristics as well as a quantitative measure. People do not merely die; they die, in each case, from some particular cause. Having observed, as it were in the gross, the quantitative changes in the deathrate as they find expression in the corresponding expectation of life, during past decades, our next inquiry, naturally, is: What, in qualitative detail, has been the nature of these changes? Have all causes been equally affected, or, since this is hardly probable, what has been the course of events as regards separate causes?

In answer let us first consider Table 8 which exhibits the twenty principal causes of death arranged in order of rank according to the number of victims which they claimed, respectively, in 1900 to 1904 and in 1925 to 1929. It requires only a glance at this table to show that fundamental changes have indeed taken place in what might be called the *internal constitution* of the general deathrate. Certain diseases which, thirty years ago, ranked high in their toll of human life, now do not enter into the accounting of the important causes at all; and conditions which now assume high positions in the list were not included at the beginning of the twentieth century. Only eleven out of the twenty diseases appear in both lists; and with the single exception of chronic nephritis, the relative rank of these is now different from that in the earlier period.

There are nine new items in the list of the first twenty causes for the years 1925-1929. They are diabetes (now ninth in rank); automobile accidents (tenth); angina pectoris (eleventh); accidental falls (thirteenth); appendicitis (fourteenth); suicide (fifteenth); congenital malformations (sixteenth); diseases of pregnancy and childbirth (seventeenth); and hernia and intestinal obstruction (eighteenth).

Of the diseases which were major causes of death thirty years ago, tuberculosis, heart disease, cancer, diphtheria, diarrhea and enteritis, and premature birth are the most noteworthy examples of change in rank.

In the first five-year period of the century, tuberculosis led all other diseases by a large margin, with a deathrate of 184.7 per 100,000. In

TABLE 8

Average Annual Deathrates per 100,000 for Leading Causes of Death, 1900-1904 and 1925-1929. Total Persons in Original Death Registration States

CAUSE OF DEATH AND RANK, 1900-1904	AVERAGE ANNUAL DEATHRATES 1900-1904	AVERAGE ANNUAL DEATHRATES 1925-1929	CAUSE OF DEATH AND RANK, 1925-1929	AVERAGE ANNUAL DEATH-RATES, 1925-1929
1. Tuberculosis—(All forms)	184.7	77.2	1. Organic heart disease[1]	219.7
2. Pneumonia—(All forms)	161.5	103.1	2. Cancer	114.3
3. Heart disease[1]	127.7	219.7	3. Pneumonia—(All forms)	103.1
4. Nephritis[2]	93.6	96.6	4. Nephritis[2]	96.6
5. Diarrhea and enteritis—(Under 2 years)	91.4*	18.0	5. Apoplexy	88.3
6. Apoplexy	75.6	88.3	6. Tuberculosis (All forms)	77.2
7. Cancer	67.5	114.3	7. Premature birth	33.4
8. Ill-defined causes	48.4	2.4	8. Influenza	25.1
9. Old age	45.1	6.5	9. Diabetes mellitus	22.6
10. Bronchitis[2]	39.3	7.7	10. Automobile accidents[5]	21.5
11. Meningitis[3]	33.1	5.6	11. Angina pectoris	19.6
12. Diphtheria	32.8	7.6	12. Diarrhea and enteritis (under 2 years)	18.0
13. Premature birth	32.6	33.4	13. Accidental falls	16.5
14. Typhoid fever	26.8	2.4	14. Appendicitis	15.2
15. Influenza	22.9	25.1	15. Suicide	14.2
16. Paralysis[4]	22.5	3.0	16. Congenital malformations	14.0
17. Congenital debility	21.8*	5.4	17. The puerperal state	12.1
18. Convulsions	19.8	1.1	18. Hernia, intestinal obstruction	10.8
19. "Other diseases of the stomach"	18.5	3.0	19. Bronchitis[2]	7.7
20. Lack of care	15.1*	0.1	20. Diphtheria	7.6

* Deathrate in Registration Area.

[1] The causes here comprised under the term "heart disease" are those of International Classification of Causes of Death, Code Number 79 of the 1900 classification, in dealing with mortality of 1900 to 1904; and Code Number 90 of the 1920 classification, in dealing with mortality of 1925 to 1929. These two combinations are not quite strictly comparable, but differ only with regard to a few relatively minor subclasses.

[2] Acute and chronic.

[3] Including epidemic cerebrospinal form.

[4] Without specified cause.

[5] Excludes deaths in collisions of automobiles with railroad trains or street cars.

the quinquennium 1925 to 1929, this disease fell to sixth in rank with a deathrate of only 77.2—not greatly in excess of one-third of the earlier figure. Organic heart disease, which was third in order at the beginning of the century, is now the leading cause of death, with a mortality almost double that of thirty years ago. In only one respect have modern sanitation and medical science been able to effect any improvement in the mortality from cardiac diseases. There has been a considerable drop in the deathrate among children and in adults up to age 45; but in older ages, the rate has greatly increased. The general aging of the population which has taken place is in part responsible for the rise in the crude deathrate for heart disease. In addition, many who, under the old conditions, would have been victims of other causes, now at older ages succumb to heart disease. This change does not, of course, represent any serious problem to the sanitarian or even to the community.

In 1900-1904, cancer was seventh on the list; now it is second, and the deathrate has increased 69 per cent. Cancer, often so terrible in its manifestations, is the disease which has shown the most extreme change for the worse during the current century. The increase in the crude deathrate is, however, due in part to the general aging of the population and in part to much improved diagnosis.

At the beginning of the century, diphtheria ranked twelfth; in 1925-1929 it stood last among the twenty leading causes. If the figures were extended to include one or two later years, diphtheria would not appear at all on the list. It is well on the road to complete elimination. What has happened with respect to this disease is an object lesson of what sanitary science can accomplish when intelligently applied.

Infantile diarrhea has dropped from fifth rank in 1900-1904, to twelfth in 1925-1929, and there has been a drop in the actual deathrate from 91.4 per 100,000 to 18.0. Progress in the control of infantile diarrhea is but a part of the general improvement in infant mortality which has been achieved.

Although there have not been large changes in the deathrates from premature birth and influenza, their relative ranks rose considerably in 1925-1929 as compared with 1900-1904. These changes in rank are due to the fact that a given numerical mortality rate now entails a much higher position in the list than the same figure would have received thirty years ago.

For certain of the causes of death which appear only in the 1925-1929 list, the deathrate has increased enormously since 1900. That for

diabetes has doubled; that for angina pectoris has tripled; and for appendicitis there has been a rise of approximately 50 per cent. Automobile fatalities were so infrequent early in the century that no enumeration was made of them at all until 1906; now they stand tenth in importance among all causes of death.

The most conspicuous example of a disease which has utterly disappeared as a leading cause of death is typhoid fever. Thirty years ago it ranked fourteenth on the list. Now typhoid fever is a vanishing disease, which in the quinquennium 1925 to 1929 took a toll of only 2.4 lives per 100,000, instead of 26.8 lives as in the first five years of the present century. The reduction in its mortality rate is one of the greatest triumphs of modern sanitary science. The problem which this disease still presents is almost entirely one of rural sanitation.

Little importance, of course, attaches to the fact that certain causes such as meningitis, "convulsions," "old age," "ill-defined causes," "congenital debility" and "lack of care" no longer appear among the leading causes, except as this indicates the progress of reporting practice in the direction of more accurate diagnosis and statement of the factors causing death.

Curtailment of Life by Different Causes

Another method of exhibiting the changes that have taken place in the qualitative composition of mortality, and one which is more closely related to the life table, consists in computing the number of years of life forfeited, on an average, to the several most important causes of death, and the changes that have occurred in these numbers. This number of years lost from a given disease depends evidently on two factors, namely, (1) on the intensity of mortality from the disease, or, as we can put it alternatively, the number of persons that die from it; and, (2) on the age incidence of the deaths. For, clearly, a disease like tuberculosis, which takes most of its victims in early adult life, does by this very fact rob them of a greater number of potential years than cancer, which has its highest mortality after midlife. It is true that of late the age incidence of tuberculosis has shifted to a later period of life. Typical childhood diseases such as measles, diphtheria and scarlet fever, may take a material toll of years in spite of their relatively low mortality, because when a very young child dies almost an entire life span is lost from the aggregate. On the other hand a disease of late life, such as cerebral hemorrhage, though characterized by a high mortality, nevertheless, does not so seriously curtail life.

TABLE 9

Numbers of Years of Life Lost from Specified Causes of Death, for Decennial Ages of Life, 1920 and 1930, Compared White Persons by Sex

(a) Death Registration States of 1920 in 1920
(b) Death Registration States of 1920 in 1930

DISEASE OR CONDITION; COLOR; SEX; YEAR		AGE								
		0	12	22	32	42	52	62	72	82
Diseases of the Heart—										
White Males.....	1920	1.79	1.95	1.93	1.95	1.96	1.91	1.75	1.37	.86
	1930	2.41	2.61	2.60	2.62	2.62	2.52	2.26	1.84	1.27
White Females.....	1920	2.00	2.10	2.08	2.09	2.07	1.98	1.81	1.42	.91
	1930	2.58	2.73	2.70	2.70	2.69	2.58	2.38	1.98	1.38
Cancer, All forms—										
White Males.....	1920	.93	1.01	1.04	1.07	1.08	.98	.74	.43	.17
	1930	1.12	1.22	1.23	1.24	1.22	1.12	.88	.53	.23
White Females.....	1920	1.50	1.60	1.64	1.70	1.62	1.33	.90	.48	.20
	1930	1.79	1.90	1.93	1.94	1.82	1.49	1.04	.59	.25
Tuberculosis, All forms—										
White Males.....	1920	1.87	1.89	1.70	1.19	.76	.43	.22	.09	.02
	1930	1.10	1.11	1.01	.76	.51	.29	.15	.06	.02
White Females.....	1920	1.90	1.87	1.48	.88	.51	.30	.17	.07	.02
	1930	1.09	1.06	.85	.51	.32	.19	.12	.06	.01
Chronic nephritis—										
White Males.....	1920	1.03	1.12	1.14	1.16	1.17	1.11	.97	.74	.46
	1930	.97	1.07	1.07	1.07	1.05	1.01	.91	.74	.48
White Females.....	1920	1.08	1.12	1.12	1.14	1.11	1.01	.86	.61	.35
	1930	1.10	1.17	1.16	1.16	1.13	1.03	.90	.69	.42
Cerebral hemorrhage—										
White Males.....	1920	.90	.98	1.02	1.06	1.11	1.10	1.01	.78	.46
	1930	.81	.90	.92	.95	.97	.96	.89	.70	.42
White Females.....	1920	1.10	1.17	1.20	1.26	1.31	1.28	1.15	.88	.53
	1930	1.04	1.11	1.13	1.16	1.18	1.13	1.03	.82	.51
Diabetes mellitus—										
White Males.....	1920	.25	.22	.21	.19	.18	.16	.12	.07	.02
	1930	.19	.21	.20	.19	.18	.17	.14	.08	.03
White Females.....	1920	.33	.29	.27	.27	.27	.24	.17	.07	.02
	1930	.37	.38	.37	.37	.38	.35	.26	.12	.03

TABLE 9—*Continued*

DISEASE OR CONDITION; COLOR; SEX; YEAR		AGE								
		0	12	22	32	42	52	62	72	82
Angina pectoris—										
White Males.....	1920	.16	.15	.16	.16	.17	.15	.12	.07	.03
	1930	.27	.30	.31	.31	.31	.27	.21	.12	.05
White Females.....	1920	.13	.08	.08	.09	.09	.09	.08	.05	.03
	1930	.14	.15	.15	.16	.16	.15	.14	.09	.04
Accidental and unspecified violence—All forms—										
White Males.....	1920	1.84	1.58	1.23	.89	.62	.41	.26	.17	.11
	1930	2.05	1.84	1.48	1.08	.79	.53	.34	.21	.15
White Females.....	1920	.73	.42	.36	.32	.28	.25	.23	.20	.18
	1930	.80	.58	.49	.43	.39	.34	.31	.28	.24

The computation of the years of life lost has been carried out for eight of the principal causes of death for white males and for white females, on the basis, first, of the life table for 1920 and the mortality for that year classified according to causes of death, and, second, on the basis of the life table for 1930 and the corresponding classified mortalities. The results of these computations, as applied to eight of the principal causes of death, are shown in Table 9 and Figure 5.

In 1920 the disease that took the heaviest toll, in years of life, was tuberculosis, 1.9 years among white persons at age zero. Among white males, accidents were a close second, with 1.8 years. Diseases of the heart also ranked high, with 1.8 years for males and 2.0 for females.

By 1930 the loss in the expectation of life at birth ascribable to tuberculosis was reduced to 1.1 years among white persons, but the loss from heart disease had gone up to 2.4 years among white males and 2.6 among white females. The loss from cancer went up from .9 years to 1.1 years among white males, and from 1.5 years to 1.8 years among white females. Further details will be gathered by inspection of Table 9 and Figure 5.

Probability of Dying from Various Causes

In still another way the life table can be made to throw light on the qualitative composition of the deathrate, and the changes in this compo-

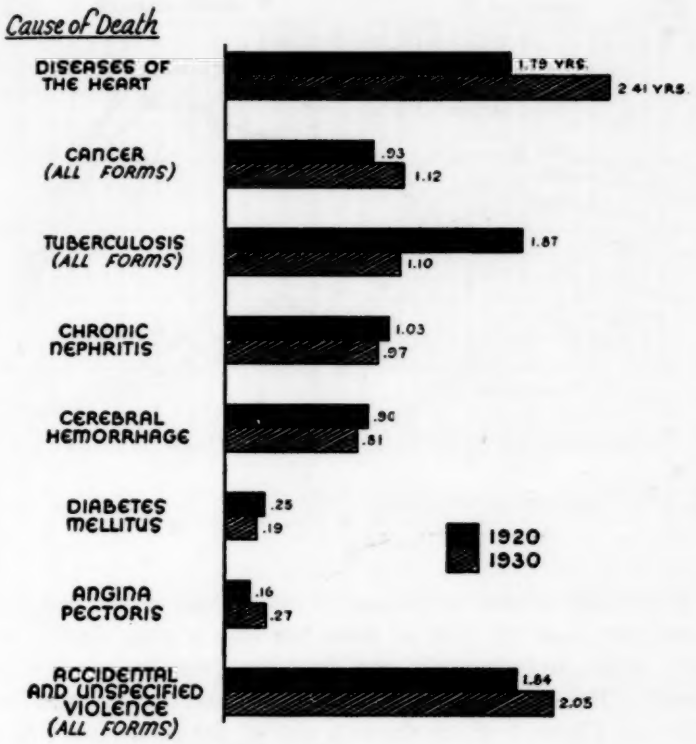


FIG. 5

sition. For, since each person must ultimately fall a prey to one or another of the multitude of different causes of death, the question arises what are the chances of eventually dying from some particular specified cause. The question can be answered by a relatively simple computation based on the life table and the observed proportion of deaths from the cause selected, among all causes, at each age of life. This computation has been carried out for several causes of death and for three separate periods, namely, 1920, 1925, and 1930, with the results shown in Table 10.

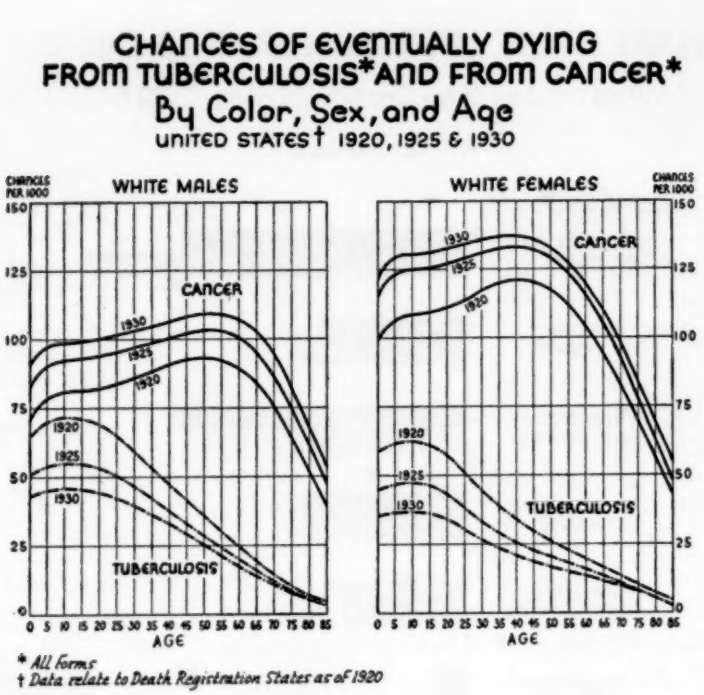


FIG. 6

Of particular interest is the case of tuberculosis and of cancer, since the deathrate from the first of these has seen a great decline in past decades, while, unfortunately, the deathrate from cancer has greatly increased. The situation with regard to these two diseases is well exhibited in Figure 6 which shows a plot of the probabilities of dying from these two diseases at successive ages of life, according to mortality conditions in 1920, 1925 and 1930. In studying the graphs shown, several characteristic features are immediately apparent to the eye. The maximum for tuberculosis comes much earlier in life than that for cancer. For white persons the three curves for the calendar years 1920, 1925 and 1930 appear one *above* the other in this order, for cancer, but one *below* the other for tuberculosis.

Another cause of death which calls for very particular mention is heart disease which today stands at the head of the list in point of the number of victims that it claims; out of every 1,000 white males born, over 200 will eventually die of heart disease under the present

TABLE 10

Chances per 1,000 of Eventually Dying from Specified Diseases or Conditions for Decennial Ages of Life United States Death Registration States of 1920 in 1920, 1925, and 1930.—White Males

DISEASE OR CONDITION; YEAR		AGE									
		0	10	20	30	40	50	60	70	80	90
Diseases of the heart	1920	146.7	167.8	171.2	178.6	188.5	198.8	211.5	219.4	216.3	180.6
	1925	179.2	200.3	203.3	209.4	217.2	227.4	238.0	249.4	252.5	237.6
	1930	203.0	222.2	225.5	231.9	240.1	251.5	265.2	281.1	292.8	284.8
Chronic nephritis	1920	88.3	101.3	104.0	108.7	114.7	120.1	124.8	128.3	125.5	109.0
	1925	95.6	107.3	109.4	112.8	116.9	122.0	127.7	133.5	130.3	109.7
	1930	91.2	100.0	101.7	104.5	108.1	113.1	119.9	128.0	130.6	116.5
Cancer, all forms	1920	69.8	80.2	82.4	86.4	90.9	93.4	89.5	76.4	51.4	33.1
	1925	82.4	92.3	94.2	97.2	100.6	103.5	100.8	86.6	61.3	35.2
	1930	90.1	98.6	100.3	103.3	106.5	109.3	107.1	94.2	67.1	44.9
Cerebral hemorrhage	1920	85.2	97.9	100.9	106.1	113.3	120.9	128.4	132.6	124.8	98.6
	1925	86.9	97.5	99.8	103.4	108.5	115.4	123.4	129.7	125.2	98.3
	1930	84.0	92.1	93.9	97.1	101.3	107.6	115.5	122.0	116.2	89.9
Tuberculosis, all forms	1920	64.6	70.8	69.5	59.4	47.7	36.1	25.0	14.8	6.7	4.6
	1925	50.9	54.8	53.7	46.6	38.0	28.7	20.2	12.1	5.5	1.7
	1930	42.7	45.3	44.6	39.6	33.0	25.3	17.5	10.7	5.0	2.0
Diseases of the arteries	1920	33.3	38.3	39.5	41.6	44.6	48.4	54.7	67.5	89.1	111.0
	1925	32.6	36.6	37.5	38.9	40.8	43.9	49.7	61.1	82.1	110.6
	1930	42.5	46.7	47.6	49.2	51.2	54.1	58.4	66.1	83.3	108.6

TABLE 10—*Continued*

Chances per 1,000 of Eventually Dying from Specified Diseases or Conditions for Decennial Ages of Life United States Death Registration States of 1920 in 1920, 1925, and 1930.—White Males

DISEASE OR CONDITION; YEAR		AGE									
		0	10	20	30	40	50	60	70	80	90
Influenza	1920	41.6	40.6	38.9	34.9	28.1	24.6	22.7	21.5	19.6	16.6
	1925	21.0	19.9	19.6	19.6	19.4	19.3	20.1	22.4	26.9	29.7
	1930	13.4	12.4	12.3	12.2	12.0	11.8	11.8	12.4	14.4	17.7
Lobar pneumonia	1920	52.8	54.9	54.2	51.5	46.8	43.3	39.7	34.9	29.8	28.6
	1925	42.1	42.6	42.4	42.0	40.6	38.0	35.3	32.3	29.8	28.8
	1930	38.5	38.4	38.1	37.7	36.4	33.9	31.2	29.1	27.4	26.5
Bronchopneumonia	1920	34.2	25.1	25.1	25.2	25.3	26.0	27.3	30.0	34.6	37.3
	1925	26.6	19.1	19.2	19.5	19.8	20.3	21.5	24.4	30.0	40.2
	1930	25.6	19.0	19.1	19.3	19.5	19.8	20.9	23.6	29.7	32.6
Angina pectoris	1920	11.2	12.8	13.2	13.9	14.7	15.2	14.8	12.6	8.5	4.6
	1925	18.4	20.7	21.2	22.0	22.9	23.4	22.6	19.1	13.2	6.4
	1930	22.7	24.9	25.4	26.3	27.2	27.6	26.4	22.5	15.6	8.3
Diabetes mellitus	1920	12.8	14.4	14.4	14.5	14.8	15.0	14.2	11.1	6.8	3.3
	1925	13.3	14.8	14.9	15.1	15.6	16.1	15.6	12.0	7.5	2.5
	1930	14.8	16.1	16.2	16.5	16.9	17.5	17.4	14.7	9.2	3.5
Diphtheria	1920	7.1	1.1	.4	.3	.2	.1	.1	.1	..	..
	1925	3.9	.6	.3	.2	.1	.1	.1	..	..	..
	1930	2.4	.3	.2	.1	.1	.1	..	..	..	..

TABLE 10—*Continued*

Chances per 1,000 of Eventually Dying from Specified Diseases or Conditions for Decennial Ages of Life United States Death Registration States of 1920 in 1920, 1925, and 1930.—White Males

DISEASE OR CONDITION; YEAR		AGE									
		0	10	20	30	40	50	60	70	80	90
Appendicitis	1920	8.8	9.2	7.7	6.5	5.2	3.9	2.5	1.5	.7	.4
	1925	10.4	10.6	9.0	7.7	6.2	4.7	3.1	1.8	1.0	.4
	1930	11.1	10.9	9.5	8.1	6.7	5.1	3.5	2.0	1.2	.5
Accidental and unspecified violence, all forms	1920	64.1	65.9	60.3	53.4	47.0	40.8	35.3	31.5	34.0	41.0
	1925	73.9	75.2	69.8	62.6	55.6	48.1	41.1	36.6	37.6	46.2
	1930	79.4	80.8	75.6	67.2	60.3	52.4	45.3	40.2	42.5	51.1
Automobile accidents including railroad and street car collision	1920	9.7	9.3	8.2	7.2	6.3	5.6	4.7	3.6	2.5	1.7
	1925	18.3	18.1	16.5	14.5	12.8	11.2	9.4	7.3	4.5	4.2
	1930	27.1	27.6	25.6	21.9	19.5	17.1	14.3	11.2	8.2	3.5
Suicides, all forms	1920	10.9	12.6	12.7	12.1	10.9	9.1	7.0	4.9	3.4	2.1
	1925	14.3	16.0	16.2	15.5	14.0	11.8	9.0	5.8	3.2	1.3
	1930	19.4	21.3	21.4	20.5	18.6	15.5	11.2	7.1	3.8	2.3
Homicides, all forms	1920	4.4	4.9	4.9	3.7	2.4	1.4	.7	.4	.3	.4
	1925	4.9	5.3	5.2	3.9	2.5	1.5	.8	.4	.1	..
	1930	4.9	5.3	5.1	4.0	2.7	1.7	1.0	.4	.2	..

TABLE 10a

Chances per 1,000 of Eventually Dying from Specified Diseases or Conditions for Decennial Ages of Life United States Death Registration States of 1920 in 1920, 1925, and 1930.—White Females

DISEASE OR CONDITION; YEAR		AGE									
		0	10	20	30	40	50	60	70	80	90
Diseases of the heart	1920	157.3	175.6	178.5	187.0	196.0	204.2	215.2	221.5	217.8	191.8
	1925	190.1	207.9	210.4	216.4	223.3	231.0	241.4	253.1	255.7	234.5
	1930	219.9	236.4	238.8	244.6	251.5	260.3	272.8	288.3	298.1	287.0
Chronic nephritis	1920	82.1	92.0	93.9	98.5	102.9	105.3	106.5	104.5	93.9	74.9
	1925	98.4	107.9	109.6	112.7	115.8	118.3	119.8	119.0	108.8	85.4
	1930	95.6	103.0	104.2	106.6	109.1	111.1	113.6	115.2	109.4	91.9
Cancer, all forms	1920	96.3	108.0	110.8	116.8	120.9	117.1	103.8	82.8	56.8	30.0
	1925	113.4	124.4	126.7	130.7	132.9	128.4	115.0	92.4	62.5	41.1
	1930	121.6	130.8	132.6	136.0	137.3	132.5	119.0	98.5	69.0	42.3
Cerebral hemorrhage	1920	99.9	112.1	115.1	121.9	129.7	136.6	142.8	146.0	138.6	104.2
	1925	102.6	112.6	114.8	119.0	124.2	129.8	136.0	140.8	137.5	106.6
	1930	102.4	110.3	112.0	115.4	119.7	124.4	129.5	134.4	129.2	106.0
Tuberculosis, all forms	1920	57.7	61.8	57.1	43.8	33.4	25.6	19.6	13.2	6.7	3.4
	1925	44.4	46.6	43.0	32.7	25.2	19.8	15.6	10.7	4.9	2.4
	1930	35.3	36.5	34.2	26.6	21.0	16.9	13.4	9.7	5.1	2.0
Diseases of the arteries	1920	28.5	32.1	33.0	35.0	37.4	40.5	45.9	56.3	75.5	100.8
	1925	29.8	32.7	33.4	34.6	36.3	38.8	43.4	53.2	72.6	102.8
	1930	36.4	39.3	39.9	41.1	42.7	45.0	49.3	57.2	74.1	101.7

TABLE 10a—*Continued*

Chances per 1,000 of Eventually Dying from Specified Diseases or Conditions for Decennial Ages of Life United States Death Registration States of 1920 in 1920, 1925, and 1930.—White Females

DISEASE OR CONDITION; YEAR		AGE									
		0	10	20	30	40	50	60	70	80	90
Influenza	1920	44.8	44.2	43.0	37.7	31.3	28.7	27.0	26.2	22.7	19.2
	1925	25.2	24.6	24.4	24.5	24.4	24.6	25.7	28.0	31.1	31.9
	1930	15.1	14.3	14.2	14.2	14.2	14.3	14.7	15.7	17.8	16.3
Lobar pneumonia	1920	48.6	49.6	49.2	46.4	42.9	41.3	39.6	37.1	32.5	25.7
	1925	35.6	35.3	35.1	35.1	34.5	34.1	33.6	32.0	28.4	24.2
	1930	33.2	32.6	32.4	32.3	31.9	31.3	31.0	29.8	27.6	22.7
Bronchopneumonia	1920	38.5	31.5	31.7	32.3	32.9	34.1	36.3	40.1	44.2	44.5
	1925	29.8	24.0	24.1	24.6	25.2	26.2	28.3	32.3	38.8	50.5
	1930	27.9	22.3	22.4	22.8	23.2	24.0	25.4	28.3	33.6	42.9
Angina pectoris	1920	7.3	8.3	8.5	8.9	9.4	9.9	10.2	9.4	7.1	3.4
	1925	11.2	12.3	12.6	13.0	13.5	14.1	14.5	13.6	10.0	5.9
	1930	13.3	14.4	14.6	15.0	15.5	16.1	16.6	15.3	11.5	7.4
Diabetes mellitus	1920	17.5	19.5	19.5	20.2	21.1	21.4	19.3	13.4	6.5	2.5
	1925	22.0	24.0	24.2	24.8	25.6	26.2	24.2	17.2	8.3	2.7
	1930	26.8	28.8	29.0	29.6	30.4	31.1	29.1	20.6	10.0	2.8
Diphtheria	1920	6.7	1.5	.6	.5	.4	.3	.1	.1	..	..
	1925	3.7	.8	.4	.3	.2	.1	.1	..	..	..
	1930	2.3	.5	.3	.3	.2	.2	.1	.1	.1	..

TABLE 10a—*Continued*

Chances per 1,000 of Eventually Dying from Specified Diseases or Conditions for Decennial Ages of Life United States Death Registration States of 1920 in 1920, 1925, and 1930.—White Females

DISEASE OR CONDITION; YEAR		AGE									
		0	10	20	30	40	50	60	70	80	90
Appendicitis	1920	6.5	6.6	5.4	4.4	3.5	2.6	1.7	.9	.5	..
	1925	7.8	7.6	6.5	5.6	4.7	3.7	2.4	1.4	.6	..
	1930	8.5	8.2	7.2	6.4	5.6	4.7	3.3	1.9	.9	1.0
Accidental and unspecified violence, all forms	1920	32.9	31.6	30.8	31.2	31.7	32.4	34.1	38.7	48.7	56.9
	1925	40.2	39.0	37.9	37.6	37.5	37.7	39.0	43.7	54.9	65.6
	1930	45.5	44.9	43.8	43.3	43.3	43.8	45.5	50.9	63.7	79.8
Automobile accidents including railroad and street car collision	1920	3.6	3.3	3.0	2.8	2.7	2.5	2.1	1.4	.8	..
	1925	7.8	7.3	6.7	6.3	6.0	5.4	4.5	3.1	1.7	.6
	1930	10.6	10.2	9.5	8.7	8.2	7.5	6.4	4.8	2.7	1.0
Suicides, all forms	1920	4.0	4.5	4.4	3.9	3.2	2.3	1.5	.8	.6	.3
	1925	4.4	4.9	4.8	4.2	3.4	2.5	1.6	.8	.4	.6
	1930	5.5	5.9	5.8	5.0	4.2	3.2	2.0	1.2	.4	.5
Homicides, all forms	1920	1.1	1.1	1.0	.6	.4	.2	.1	.1	.1	..
	1925	1.3	1.3	1.2	.8	.5	.3	.2	.1	.1	..
	1930	1.5	1.5	1.3	1.0	.6	.4	.2	.1	.1	..
Puerperal state	1920	18.6	21.0	19.9	11.3	2.4	..	..	..	..	..
	1925	14.1	15.5	14.5	8.5	1.8	..	..	..	..	..
	1930	12.1	13.0	12.2	7.0	1.5	..	..	..	..	..

conditions of mortality; that is, approximately one in five. The chances (at birth) are in fact greater of eventually dying from heart disease, than from any other single cause, and even if we add together the chances of dying from two such important causes as tuberculosis and cancer, the sum for these two is actually less than the chances of dying from heart disease. For according to mortality conditions as of 1930, the probability, at birth, of eventually dying from tuberculosis, among white males, is 43 per thousand, from cancer, 90 per thousand; so that the chances of eventually dying from either one or the other of these two causes are 133 per thousand, and less than the chances of dying from "heart disease," namely, 203 per thousand. The corresponding probability figures for white females are tuberculosis 35, cancer 122, summing up to 157 per thousand, as against 220 per thousand for heart disease.

At successive ages of life, the probability of dying from heart disease increases steadily among white persons until the neighborhood of the eightieth year of life, after which it decreases again.

A comparison of the figures for the years 1920, 1925 and 1930 shows quite clearly that the chance of dying from heart disease has shown a steady increase in these two quinquenniums. This is in part merely the inevitable accompaniment of the decreasing proportion of deaths from those causes over which we have gained control. However, as we know from a study of the age-specific deathrates, there is also a definite increase in the mortality from diseases of the heart after age twenty-five for white males, and after age forty-five for white females.

Another interesting feature exhibited in the table is the fact that the probability of dying from heart disease, in the white population, at any rate, is greater for females than for males. This is consistently the case in the three calendar periods examined. The difference is not very great and is due probably, in whole or in part, to the familiar fact that women are longer lived than men, and that, therefore, there must be a relative predominance of deaths among women from causes characteristic of the older ages, and among these organic heart disease is preeminent.

Among heart conditions not included in the general classification of organic heart disease one calls for special mention here because of the very marked increase which it has shown in recent years, namely, angina pectoris. It is, of course, possible that a part of this increase is due to a sort of medical *fashion,* which for some reason has favored

a diagnosis of angina pectoris in recent years. The fact, however, that the increase has been very definite and rather systematic within the last ten years when taken in groups of five years, rather points to the fact that there must be some real significance in this increase. At birth the probability of eventually dying from angina pectoris for white males was 11.2 in 1920, 18.4 in 1925 and 22.7 in 1930. Thus in the decennium the figure has almost exactly doubled. Among females the situation is similar, except that the probabilities are individually markedly lower than among males as follows: in 1920, 7.3; in 1925, 11.2; and in 1930, 13.3. Here again there is an approximate doubling in ten years.

Another disease for which the figures exhibited in the table show an increase in successive quinquennia is diabetes. For white males at birth, the probabilities of eventually dying from this disease were as follows: in 1920, 12.8; in 1925, 13.3; and in 1930, 14.8; for white females in 1920, 17.5; in 1925, 22.0; and in 1930, 26.8.

Important causes of death in later life are cerebral hemorrhage and chronic nephritis. As regards cerebral hemorrhage, at age zero and age ten there is relatively small change of any kind shown in the probability of dying from this disease. From age twenty on there is a tendency for a not very marked diminution. This statement holds for both white males and white females. The figures for nephritis are somewhat similar in character, but differ in one respect, namely, that the year 1925, at practically all ages shown in the table, stands somewhat higher than either 1920 or 1930. The difference is hardly significant.

That the chances of eventually dying from diphtheria, figured at birth, should have been reduced by about two-thirds in the decennium, as will be seen from an inspection of the table, will not come as a surprise.

Among causes of death other than from disease, two stand out with unfortunate prominence by the increases shown in the probability of dying therefrom. The first is automobile accidents, for which the probability at birth, in 1930, namely, 27.1 per 1,000 among white males, was fifty per cent higher than in 1925, an ominous increase in so short a period. Secondly, the probability at birth of eventually dying by suicide has gone up among white males from 10.9 in 1920 to 19.4 in 1930, almost 100 per cent in ten years. This comparison is perhaps not entirely fair since the year 1930 is affected by the economic depression.

Among the causes of death in women the conditions associated with the puerperal state naturally invite special attention. At birth the probability of eventually dying from these causes was 18.6 per 1,000 in 1920, fell to 14.1 in 1925, and to 12.1 in 1930. This decrease can hardly be accepted with whole-hearted satisfaction; it is due undoubtedly in large part to the rapidly falling birthrate.

V

COMPARISON WITH FOREIGN COUNTRIES

We have noted in preceding sections that the United States has made a favorable record in the past in the reduction of mortality in early childhood. Comparison of our infant mortality with that among some of our contemporaries in Europe gives us cause for satisfaction in this respect. We are doing somewhat better than England, and markedly better than Germany, as is shown in the following figures:

	1000 q_0	
	Males	Females
England and Wales 1921-1922	89.96	69.42
United States 1919-1920 (white).....	84.33	67.32
Germany 1924-1926	115.38	93.92
United States 1925*	75.14	59.29

While the deathrate in the first year of life appearing in the German life table very materially exceeds those in the table for the United States, this relation does not hold throughout life. So, for example, in the series of quinquennial values of q_x for ages 2, 7, 12 ...92 we find lower values in the German table (1924-1926)† for males than in the American (white males 1925) except at ages 22, 27 and from 72 to the end of life, where the American figures are somewhat lower. Even if we compare the 1930 American table with the 1925 German table, there are still many ages at which the German table shows more favorable mortality than the American, namely, in the range from five years to 15 years and from 31 years to 67 years. Yet the infant mortality in Germany in 1925 was so high as to depress the expectation of life at

* Life table prepared by the Statistical Bureau of the Metropolitan Life Insurance Company. Recomputed after data of 1930 census became available.

† Statistik des Deutschen Reichs-Vol. 360: Die Bewegung der Bevölkerung. Deutsche Sterbetafeln 1924-1926, p. 168, Berlin, 1930.

birth in 1925 (namely 55.97 for males) below that for the United States in either 1930 or 1925 (namely 59.09 or 57.88, respectively, for white males).

This comparison of our record with those of our contemporaries abroad very clearly indicates that as regards mortality in adult life we fall short, not only of ultimate possibilities, but even of actual accomplishment in other countries. This fact has a direct bearing on the topic to which we now, in conclusion, turn our attention, namely, our ultimate prospects of longevity.

VI

PROSPECTS FOR FUTURE IMPROVEMENT

In a previous section a table was given exhibiting the number of years of life sacrificed to each of the most important causes of death, in 1920 as contrasted with 1930. The gains thus shown, for certain diseases and ages of life, suggest the question: How much further may we expect the improvement to go? What may we reasonably look for as the future average length of life? Some writers have expressed themselves in unbounded optimism on this point, but a thoughtful consideration of the facts does not justify such extreme optimism, at least in the present state of our knowledge and of the evolution of the human organism. The 1.1 years of life still being lost per person, in the white population, through the ravages of tuberculosis, we may indeed, hope to see to a large degree deleted from the vital account of the future population, for the mortality from this disease has for many years past declined rapidly and steadily. These gains are the more fortunate as tuberculosis attacks largely persons in early adult life, just at the time when they promise to be most valuable to society. A life saved at this period is worth much not only in mere years for the individual, but also in the measure of his contribution to the life of the race.

With regard to certain other groups of causes, too, we may be optimistic; among these a most serious group are the diseases and abnormalities of early infancy. Here the remedy will lie chiefly in prenatal care for the expectant mother. Good progress has been made in this field, but much remains to be done. Any gains that we shall register here are of particular importance, because an infant saved means many potential years of life saved.

The picture is different when we come to the causes of death characteristic of the later ages. Here gains have been small, or there may have been actual losses in the recent decade, in the United States. At the present time it would seem that we are here dealing, if not with an unconquerable foe, at any rate with a foe unconquered. Unqualified pessimism, however, would even here be out of place. In the case of cancer, at least, we possess knowledge which, properly applied, should help to reduce mortality. The facts, unfortunately, do not as yet bear out this hope; in spite of the active fight against this disease, so far the deathrate from it has been on the upgrade in recent years, and it would seem that this remains true even after allowance is made for changes in age composition, for better reporting and for improvement in diagnosis, and hence in the classification of deaths by cause. We are dealing here with unsolved problems.

Hypothetical Life Table

Guided by past experience, by what has been accomplished, and a knowledge of where we have so far failed, we may briefly consider the somewhat speculative question of the ultimately attainable expectation of life—ultimately, that is, in the light of present knowledge, and without intervention of radical innovations or fantastic evolutionary change in our physiological make-up such as we have no competent reason to assume.

A hypothetical life table prepared by the senior author some years ago, to represent such a prospect for the future, has already proved too conservative. At a time when the actual length of life was about 57 years, this hypothetical table promised an ultimate figure of 64.75 years. Since then this hypothetical figure has been actually exceeded in New Zealand as far back as 1921-1922 and is over three years below the expectation of females in that country in 1931. It is surely reasonable to expect that what New Zealand is achieving at present we may hope to exceed in due course. Accordingly a new hypothetical life table has been constructed which may be found useful as a basis for various computations.

This life table is based fundamentally on the New Zealand life table for females 1931. With this as a background, a series of values of q_x for each age of life have been estimated, using our best judgment of what we thought could reasonably be expected in the United States as an ultimate achievement. In selecting these figures we have had the

guidance, not only of the New Zealand table, but also of our own 1930 table and the experience of the Metropolitan Life Insurance Company among some of the more favored classes of policyholders. It is felt that what can today be achieved among the more favored classes, should be attained in the near future by the average man, and from this standpoint we believe that the figures we have adopted are essentially reasonable.

Naturally, some of the greatest gains from the current mortality in our country must be expected at the early childhood ages, where there is still considerable opportunity for improvement. Here we have assumed that ultimately a q_x of 25 per 1,000 may be attained at age zero instead of a value of about sixty which now prevails. In New Zealand they had already reached the low figure of 25.5 for females in 1931. At age one we have assumed a reduction of ten per cent from the mortality observed among females in New Zealand, 1931. For each succeeding age after age one, we have taken an additional quarter of one per cent from the mortality as shown for New Zealand females, 1931, until at age twenty-one, a fifteen per cent reduction was obtained. It was then assumed that this reduction of fifteen per cent from the mortality of the table considered so far is attainable for all succeeding ages to seventy-four, after which the reduction was decreased by one per cent for each subsequent age until it was only ten per cent at age seventy-nine. For ages from 80 to 88, our hypothetical mortalities are guided by those observed for New Zealand, 1931, and United States white females, 1930. Mortalities for the remaining ages of life were based on those observed in the last-named table. The factors we have applied at successive quinquennial ages of life are shown in the fourth and fifth columns of Table 11. In no instance has there been assumed a reduction of more than fifteen per cent from an observed mortality. We feel that we have not unreasonably trimmed down the values of q_x from those observed in the general population, especially in the light of the experience among Ordinary policyholders of the Metropolitan Life Insurance Company, which is not far removed from the conditions we have assumed.

From the values of q_x obtained by applying the factors indicated, the new hypothetical life table exhibited in Table 11 and Figure 7 has been constructed. Its relation to certain actual life tables is immediately seen on inspection of the chart Figure 7. In this chart the lower panel exhibits the curves for the expectation of life, e_x, at successive

TABLE 11

Hypothetical Life Table—Representing Ultimate Longevity Attainable Under Modern Medical and Sanitary Science

AGE x	NUMBER ATTAINING AGE x OUT OF 100,000 BORN ALIVE	PROBABILITY PER 1,000 OF DYING WITHIN ONE YEAR AFTER ATTAINING AGE x			AVERAGE LENGTH OF LIFE
			Ratio to corresponding item in		
	SURVIVORS l_x	Probability $1{,}000q_x$	New Zealand (1931) females	U. S. Reg. States (1930 in 1930) wh. females	EXPECTATION OF LIFE $\mathring{e}_x$
0	100,000	25.00	.981	.516	69.93
1	97,500	3.34	.900	.405	70.71
2	97,174	1.97	.897	.436	69.95
3	96,983	1.82	.895	.581	69.09
4	96,806	1.73	.892	.727	68.22
5	96,639	1.50	.890	.754	67.34
10	96,144	.56	.875	.519	62.67
15	95,843	.99	.860	.615	57.85
20	95,221	1.61	.850	.599	53.21
25	94,369	2.18	.850	.663	48.66
30	93,216	2.80	.850	.773	44.23
35	91,858	3.09	.850	.741	39.83
40	90,438	3.15	.850	.602	35.41
45	88,875	4.31	.850	.626	30.98
50	86,698	5.80	.850	.618	26.69
55	83,876	8.32	.850	.610	22.50
60	79,645	13.83	.850	.673	18.55
65	73,240	21.05	.850	.679	14.94
70	64,508	32.69	.850	.683	11.60
75	52,327	56.65	.860	.762	8.69
80	36,391	91.15	.903	.796	6.38
85	19,914	152.56	.894	.916	4.60
90	7,546	212.96	...	.929	3.44
95	1,861	295.31	...	.959	2.50
100	237	406.54	...	.993	1.78

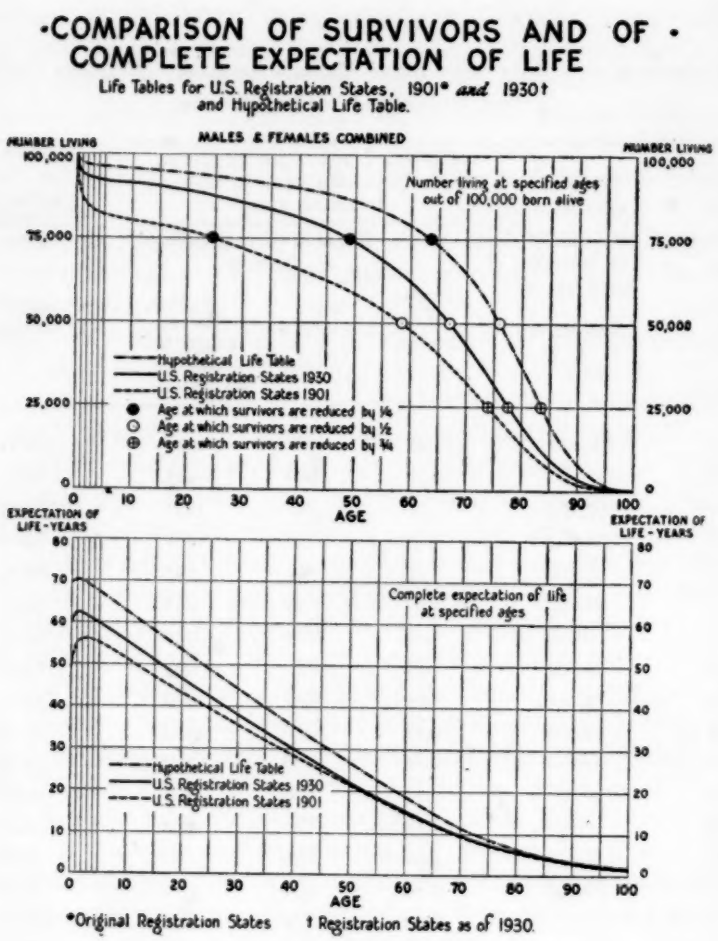


FIG. 7

ages, according to three life tables, namely, (1) that for the United States Original Registration States, (total persons) 1901; (2) that for the United States Registration States, total persons (1930); (3) the new hypothetical life table. The greatest difference between the three curves is found, naturally, at age zero, that is, at birth. At this point it will be seen that the 1901 table gave an expectation of life (mean length of life) of 49.24 years. The 1930 table betters this by over ten

years, giving an expectation of 59.70. The hypothetical table promises an ultimate expectation of life at birth of 69.93 years, or to speak in round numbers, 70 years, the biblical three-score and ten. These gains, judging by all present signs, must be regarded as about the limit of what we may reasonably hope to attain by the application of modern medical practice, public health methods and sanitation. More than this we could achieve only if radically new discoveries should be made either toward the care or toward the prevention of some of those diseases which today prove highly refractory to all medical effort. It is true that such discoveries are entirely within the range of possibility, but we have no sufficient grounds for taking them for granted, nor should we have any guide, once we entered the domain of pure conjecture, to set any bounds whatsoever to our speculations.

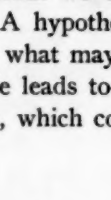
The upper panel of the chart Figure 7 shows the curve of survivors l_x to each decennial age out of 100,000 born, according to the same three life tables. The eye immediately grasps the general situation represented by these curves. A feature of particular interest, which may here be singled out for special attention, is the change that has taken place, and the further change anticipated by the hypothetical table, in the characteristic age at which a cohort of 100,000 persons born is reduced to ¾, ½ and ¼ of its original number. The points on the curve at which these events occur are identified by black, white, and crossed circles respectively. The greatest change has taken place in the age at which the cohort is reduced to ¾ of its original number. This happens at the age of 24, 49 and 63 respectively according to the three life tables. These ages stand in the ratio of 1 to 2 to 2.6. The half-way points lie closer together, namely, at ages 58, 67 and 76, a gain of nine years at each step. Lastly, the age at which only ¼ of the cohort is left surviving, is found at 74, 78 and 83 years respectively, so that even here there will still be a gain of nearly ten years over 1901 when conditions represented by the hypothetical table are established. The pessimist might urge that this is not an unmixed blessing. It means a weighting of the future population with more old people, many of whom cannot be self-supporting. At any rate the problem of providing for the care of the aged, which is pressing enough now, will not abate when the average length of life is extended by ten more years, unless in the meantime we shall have established an adequate system for dealing with this very serious issue.

VII

SUMMARY AND CONCLUSION

Looking back to the beginning of our existence as an independent nation we have every reason to be gratified by the large gains in longevity that we were able to record in the past 100 years, and more particularly in the last forty years. This is an achievement which probably even a confirmed optimist would hardly have been sanguine enough to foresee in anticipation. Undoubtedly the position gained must be credited very largely to the account of modern organization of public health service, with all its ramifications. It has freed us from the threat of typhoid fever, it has placed in our hands highly efficient control of malaria, it has made diphtheria a wholly preventable cause of death, it has immensely reduced our infant mortality, and to it also must be ascribed largely our winning fight over tuberculosis. The most effective measures have undoubtedly been the preventive measures, but we should be singularly in error if we should forget to mention also the innumerable lives that have been saved by the success of modern medical and surgical practice which annually save many lives by therapeutic and operative means. Nevertheless, those who interest themselves in the health program from a demographic standpoint will probably be inclined to emphasize the utility of preventive measures on the old but not worn out principle that prevention is better than cure.

The outlook for further improvement in the average length of life is entirely favorable, since we have not yet fully availed ourselves of the knowledge which we now possess towards reducing mortality. At the same time there is a limit to what we can hope to accomplish on the basis of existing knowledge. A hypothetical life table constructed to represent as nearly as possible what may reasonably be expected to be achieved in prolongation of life leads to a figure of seventy years for the expectation of life at birth, which coincides with the biblical three score years and ten.



MORTALITY IN NEW YORK CITY A CENTURY AND A QUARTER AGO*

BY MARY DALTON

ONE of the longest series of annual mortality statistics available in the United States is that for the city of New York beginning with the year 1804. Records for two other cities, Baltimore and Boston, go back nearly as far but do not include details as to sex, age, and cause of death. For Baltimore we have the total of deaths and deaths from various causes from the year 1812 (1), but for the early period subdivisions by sex, age, and color are given for certain years only. The Boston statistics begin with 1811 (2), but are not classified by cause and age until many years later.

The "Return of Deaths in the City of New York for the Years 1804, 5, 6, 7, and 1808" is probably the earliest printed record of deaths in the city. A century and a quarter ago the city occupied only a fraction of the vast area of the present Greater New York and very likely did not extend much beyond the City Hall Square, its northern boundary in 1790 (3). The eight-page report contains three tables and some interesting short comments. The first table shows, for each of the years 1805-1808, the number of deaths of "men," "women," "boys" and "girls" by months; the second, deaths by age for the same years. The third table gives the number of deaths from specified causes in each of the five years 1804-1808.

CAUSES OF DEATH

The simple alphabetical list of causes of death is in interesting contrast to the International Classification now in use. There are many general titles in the old list which are now included under other, more specific causes. In 1804 there were, for example, 5 deaths from "cholic"; "cold" was responsible for 18 deaths; "cramp in the stom-

* From the Division of Vital Statistics, New York State Department of Health, and the Department of Biostatistics, School of Hygiene and Public Health, The Johns Hopkins University, No. 184.

ach," 3; "decay," 43; "hives," 75; "mortification," 9; "sore throat," 10; and "teething," 33. In 1805, 11 deaths were charged to "drinking cold water," and 4 persons died from being "frost bitten." "Nervous head ach" was the cause of 2 deaths in 1807 and "caries" caused 1 death in 1808. Approximately 100 deaths each year were charged to "fevers," subdivided into ten different types—bilious, hectic, inflammatory, intermittent, malignant, puerperal, putrid, remittent, scarlet, and typhus.

The remarks of the City Inspector, John Pintard, on certain causes of death and the accuracy of reporting fill the last two pages of the report. A few are quoted below, including a reference to "consumption" which sounds familiar to modern ears:

Consumption. The amount of deaths under this head must be accepted with considerable allowance; as there is just reason to conclude that many are reported having died of *consumption,* from the difficulty of ascertaining the precise malady. With every abatement, however, it is a very alarming disease, incidental in some degree to our latitude, but undoubtedly aggravated by the imprudent adoption of *Fashions in Dress* adapted to more genial climes.

Drowned. With the increase of our population and navigation, this head progressively increases.

Infanticide. In a country, where so little pretext is afforded for the commission of this horrid and unnatural crime, it is to be lamented that any instances should occur.

Smallpox. The extension of VACCINATION will, it is confidently hoped, eventually exterminate this once afflicting scourge. The number of instances have, however, increased, the last year, which ought to attract the attention of the Faculty.

Suicide. Instances still occur of this *most awful crime.*

In conclusion, Mr. Pintard states:

. . . . the reports of deaths in this city and county are perfectly accurate. Stricter attention ought to be paid by physicians, in giving their certificates of the diseases, of which their patients died.

Should the Corporation persevere in enforcing accurate returns of deaths, the bills of mortality will, in a series of years, become incalculably useful, not only to medical professors, but for various other important purposes.

The Annual Report of the City Inspector for the year 1853 contains a "Table of the Semicentennial Mortality . . ., Comprising the Full Period from January 1, 1804 to December 31, 1853 inclusive," which gives, in addition to the information outlined above for 1804-1808, the deaths by sex and by age for 1804. The list of causes in the semicentennial table shows considerable elaboration and improvement in classi-

fication. The causes are arranged in two large classes, each of which is subdivided into several groups, as follows:

Class No. I. *Zymotic, Epidemic, Endemic and Contagious Diseases*
- Brain and Nervous System
- Digestive System
- Generative System
- Integumentive System
- Respiratory System
- Uncertain, Indefinite

Class No. II. *Sporadic Diseases*
- Brain and Nervous System
- Circulatory System
- Digestive System
- Generative System
- Integumentive System
- Locomotive and Osseous System
- Old Age or Decay
- Premature Birth
- Respiratory System
- Stillborn
- Urinary System
- Uncertain or Indefinite
- Unknown

In the early years statistics of deaths were taken from cemetery records (4). In 1853, according to the report for that year (5), the passage of the Amended Registry Act (April 2) and the establishment of the Bureau of Statistics and Sanitary Inspection were "fruitful of much important utility in the accomplishment of the wise purposes sought in the institution of health-promoting measures." In addition to the summary for the fifty-year period previously mentioned, more detailed tables for the year were published than heretofore. But according to the Report of the Metropolitan Board of Health for 1866 (4), although the Act of 1853 provided "specific and stringent regulations" for registration, "serious irregularities prevailed" until the adoption of new regulations in the Spring of 1866. An order directing the establishment of a Bureau of Vital Statistics was passed by the Metropolitan Board of Health at its first meeting for the year, March 5, and "the archives hitherto pertaining to the office of the City Inspector were transferred to the new Bureau."

Another change came in 1898 (6) when, under a new charter, various towns and cities were consolidated into Greater New York, con-

sisting of five boroughs, with an Assistant Registrar of Vital Statistics in each. The old city, as it existed before January 1, 1898, was divided into the Borough of Manhattan and the Borough of the Bronx, the city of Brooklyn became the Borough of Brooklyn, several towns and villages of Queens County formed the Borough of Queens and Richmond County (comprising Staten Island) became the Borough of Richmond. The vital statistics records in Manhattan, the Bronx, and Brooklyn are comparable with those for the City of New York and the city of Brooklyn before 1898. Comparison with earlier years, however, cannot be made in the Boroughs of Richmond and Queens.

LIFE TABLES

As a means of measuring the changes in longevity of the population of the City of New York in a period of a hundred and twenty-five years, two life tables were computed, one based upon the earliest and the other

TABLE 1

Abridged Life Table for New York City, 1805-1808 *

AGE INTERVAL	OF 100,000 ALIVE AT AGE 10		RATE OF MORTALITY PER 1,000	EXPECTATION OF LIFE
Period of lifetime between two exact ages	No. alive at beginning of age interval	No. dying in age interval	No. dying in age interval among 1,000 alive at beginning of age interval	Average length of life remaining to each one alive at beginning of age interval
x to $x+10$	l_x	d_x	$1000q_x$	$\mathring{e}_x$
Years			*Annual rate*	*Years*
10- 19	100,000	6,582	65.82	37.64
20- 29	93,418	15,099	161.63	29.84
30- 39	78,319	17,726	226.34	24.57
40- 49	60,593	15,791	260.61	20.28
50- 59	44,802	15,012	335.08	15.70
60- 69	29,790	14,872	499.23	11.10
70- 79	14,918	10,820	725.29	7.30
80- 89	4,098	3,810	929.61	4.48
90- 99	288	282	980.37	2.66
100-109	6	6	1000.00	2.50

* Based upon the annual average number of deaths recorded in 1805-1808 and the population estimated as of January 1, 1807.

upon the latest data available. The tables were constructed by a method for abridged life tables developed by Dr. Lowell J. Reed and Miss Elizabeth Parkhurst at the Johns Hopkins School of Hygiene and Public Health. The table for 1805-1808 (Table 1) is based upon the annual average number of deaths in the old City of New York and the population estimated as of January 1, 1807. For the later table (Table 2) the average number of deaths for the years 1929, 1930, and 1931 within the present city limits, and the population estimated as of July 1, 1930 were used. Since the censuses of 1800 and 1810 do not show subdivisions of the population under 10 years of age, it was impossible to compute specific death rates at these ages. Both life tables are, therefore, confined to persons 10 years of age and over.

The largest percentage decrease in mortality from the early to the later table is exhibited at 20-29 years, where the rate was 79 per cent lower in 1929-1931 than in 1805-1808. The decrease of 77 per cent in the group 30-39 was next in order, followed by a drop of 72 per cent

TABLE 2

Abridged Life Table for New York City, 1929-1931 *

AGE INTERVAL	OF 100,000 ALIVE AT AGE 10		RATE OF MORTALITY PER 1,000	EXPECTATION OF LIFE
Period of lifetime between two exact ages	No. alive at beginning of age interval	No. dying in age interval	No. dying in age interval among 1,000 alive at beginning of age interval	Average length of life remaining to each one alive at beginning of age interval
x to $x+10$	l_x	d_x	$1000q_x$	$\mathring{e}_x$
Years			*Annual rate*	*Years*
10-19	100,000	1,834	18.34	53.59
20-29	98,166	3,263	33.24	44.48
30-39	94,903	4,884	51.47	35.83
40-49	90,019	9,027	100.28	27.48
50-59	80,992	16,619	205.20	19.92
60-69	64,373	24,475	380.21	13.67
70-79	39,898	24,436	612.45	8.91
80-89	15,462	13,382	865.50	5.39
90-99	2,080	2,080	1000.00	2.37

* Based upon the annual average number of deaths recorded in 1929-1931 and the population estimated as of July 1, 1930.

at ages 10-19 and 62 per cent at ages 40-49. After 40, the percentage decrease becomes steadily smaller with age, except at 90-99 where an increase in rate was recorded. However, the increase has little significance since the figures in this age group were comparatively small and undoubtedly not very accurate.

An increase in the expectation of life is, of course, a result of the lower death rates. At age 10 the expectation of life increased from 37.64 to 53.59, or nearly sixteen years. The greatest percentage increase (49 per cent) occurred at age 20, corresponding to the greatest decrease in the death rates, with an increase in expectation of life (14.64 years) almost as great as at age 10. The expectation at age 30 also increased by a higher percentage (46 per cent) than at age 10. The percentage increase at age 10 was third in order of size, followed by percentages which decrease steadily with age for those 40 years of age and over.

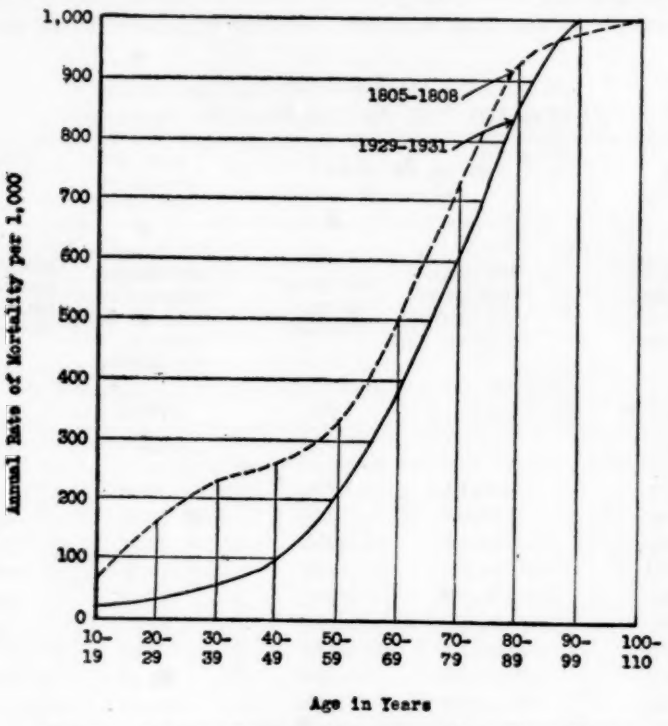


FIG. 1. ANNUAL RATE OF MORTALITY, NEW YORK CITY, 1805-1808 AND 1929-1931

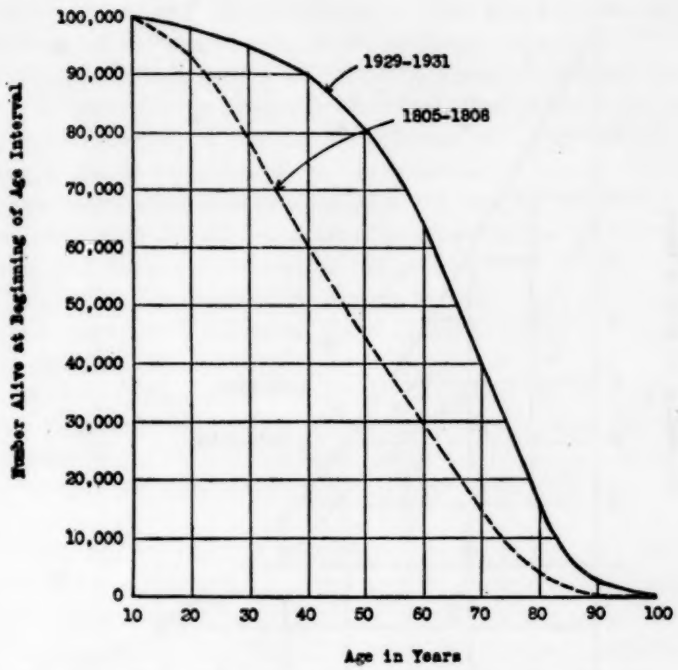


FIG. 2. NUMBER OF SURVIVORS OUT OF 100,000 ALIVE AT AGE 10, NEW YORK CITY, 1805-1808 AND 1929-1931

The number of survivors was one-half the original number at an age between 40 and 50 years in 1805-1808, while in 1929-1931 four-fifths survived to age 50 and one-half to an age between 60 and 70.

The mortality, number of survivors, and expectation of life at various ages in the two periods are illustrated in Figures 1, 2, and 3.

The difference in the form of the two mortality curves in Figure 1, at ages under 50, is very striking. The unusually sharp rise at the beginning of the curve for 1805-1808, resulting from the high death rates in the first four age groups, leads one to question the reliability of the data. There are indications, however, that the rates at these ages actually were very high. The number of deaths was undoubtedly even greater than the number recorded and it is not likely that the population was under-enumerated to a greater degree in these age groups than in the others. The possible error in estimating the age distribution of the colored population, which was not given in the censuses of

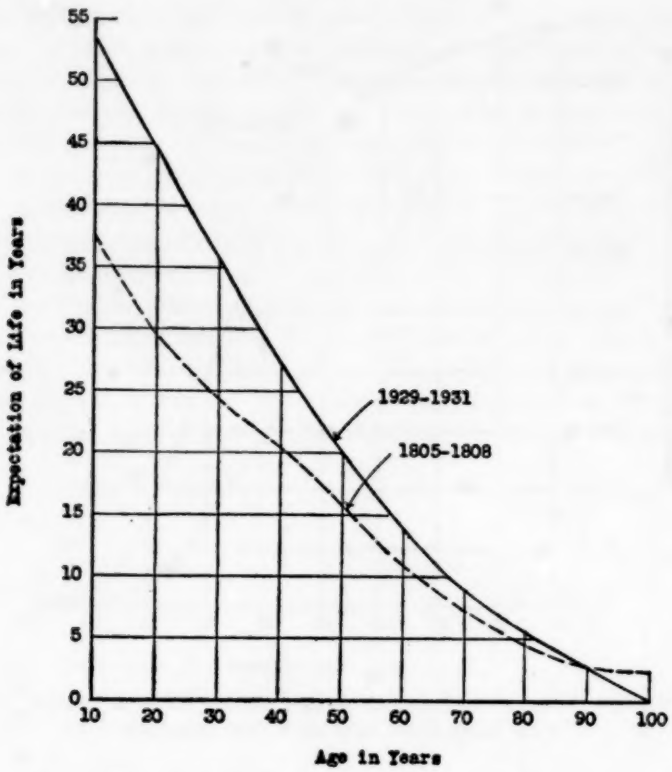


FIG. 3. COMPLETE EXPECTATION OF LIFE, NEW YORK CITY, 1805-1808 AND 1929-1931

1800 and 1810, was also too small to change the general configuration of the curve. On the other hand, it is well known that in recent years most of the decline in the general death rate has resulted from decreases in the mortality of infants, children, and young adults, while mortality from diseases of older age, such as diseases of the heart and arteries, cancer, and diabetes, has apparently increased.

In New York City the death rate from all causes decreased from 26.7 per 1,000 population in 1805-1808 to 11.0 in 1929-1931. At the same time mortality from pulmonary tuberculosis, an important cause of death in young adult life, dropped from 511.3 per 100,000 population to 66.1. In 1805-1808 nearly 20 per cent of all deaths were charged to "consumption," while in 1929-1931 the same cause was responsible

for only 6.0 per cent. It is difficult to make a comparison of deaths from other causes because of changes in classification.

Since the age distribution of deaths by cause is not given for 1805-1808, it is impossible to determine the exact effect of the high mortality from tuberculosis upon the curve in Figure 1. The annual average number of deaths from this cause (427) was large enough, however, so that if we assume that most of them occurred between the ages of 20 and 50 and recompute the mortality per 1,000 alive at the beginning of the age intervals, the curve approaches the general shape of that for 1929-1931; similarly, the curves for the early years in Figures 2 and 3 approach more nearly the usual shape of survivorship and expectation of life curves.

It appears from comparison of the life table for New York City in 1805-1808 with the Carlisle life table (7) and a table for towns in Massachusetts and New Hampshire computed from records on file in 1789 (8) that the rates of mortality in New York City were relatively high. (Table 3)

The Carlisle life table is based upon deaths in the nine years 1779 to 1787 in the parishes of St. Mary and St. Cuthbert in Carlisle, England and the population returns of the censuses taken as of January 1, 1780 and December 31, 1787. The table for Massachusetts and New Hampshire includes a total of 4,893 deaths reported from various towns over a period of years. In the latter table, however, the death rates were too high and the expectation of life too low, especially in the younger age groups, due to the assumption that births and deaths were equal when the annual number of births actually was double the number of deaths.

Although the higher mortality in New York City may be partly due to incomplete enumeration of the population, a greater part of the difference is probably a result of more favorable living conditions in Carlisle, a small town in England, and in rural areas of Massachusetts and New Hampshire. Statements from the City Inspector's "Annual Report of Deaths in the City and County of New York, for the year 1834" (9), although written thirty years later, probably apply as well to conditions in 1805-1808:

> Some cause should be assigned for the increase of deaths beyond the increase of the population, and none appears so prominent as that of intemperance, and the crowded and filthy state in which a great portion of our population live, and apparently, without being sensible of their situation; and we have serious cause to regret

that there is in our City so many mercenary landlords, who only contrive in what manner they can stow the greatest number of human beings in the smallest space.

There is another cause that may have a tendency to produce disease, and consequently increase the number of deaths, that is the impure water that many of our population have to use—although most of the wells of our City when first dug, produced good water, they have become from the filth that must necessarily filter into them from the streets, especially in the vicinity of cowstables, slaughter houses, dye establishments and factories, very unwholesome.

At the same time conditions in the small towns for which the other life tables were made were unusually favorable. The expectation of life for persons 40 years of age and over in the Carlisle table was greater

TABLE 3

Annual Rates of Mortality and Expectation of Life from Life Tables for New York City, 1805-1808; Carlisle, England, 1779-1787; and Massachusetts and New Hampshire, 1789 *

AGE INTERVAL	RATE OF MORTALITY PER THOUSAND			EXPECTATION OF LIFE		
Period of lifetime between two exact ages	Number dying in age interval among 1,000 alive at beginning of age interval			Average length of life remaining to each one alive at beginning of age interval		
	N. Y. City 1805-1808	Carlisle 1779-1787	Mass. and N. H. 1789 *	N. Y. City 1805-1808	Carlisle 1779-1787	Mass. and N. H. 1789 *
x to $x+10$	$1000q_x$			$\mathring{e}_x$		
Years	*Annual rate*			*Years*		
10- 19	65.82	57.28	128.91	37.64	48.82	39.23
20- 29	161.63	73.56	170.40	29.84	41.46	34.21
30- 39	226.34	100.50	186.03	24.57	34.34	30.24
40- 49	260.61	133.60	193.49	20.28	27.61	26.04
50- 59	335.08	171.48	209.63	15.70	21.11	21.16
60- 69	499.23	340.93	314.34	11.10	14.34	15.43
70- 79	725.29	603.08	532.95	7.30	9.17	10.06
80- 89	929.61	851.00	840.49	4.48	5.51	5.85
90- 99	980.37	936.62	1000.00	2.66	3.28	3.73
100-109	1000.00	1000.00	.....	2.50	2.28	...

* The life table for Massachusetts and New Hampshire is based upon records which were collected from various towns over a period of years and were in the files of the American Academy of Arts and Sciences in 1789.

than in New York City in 1930, and that for persons 10, 20, and 30 years of age was nearly as great. Similarly, in the table for towns in Massachusetts and New Hampshire the expectation of life at ages 50 and over was greater than that at the same ages in New York City in 1930. That these differences were not simply a result of high mortality in New York City is shown by the fact that the expectation of life at some ages in the Carlisle table is greater than that in many of Glover's life tables for 1910 (10)—greater than for males in Boston, Chicago, New York City and Philadelphia at 10 years of age and over, in New York State at 20 years and over, New Jersey at 30 years and over, Massachusetts at 40, Michigan at 70 and Indiana at 80 and over. It is also higher than that for both sexes in the entire original death registration area of the United States in 1910 at ages 50, and 70 years and over, and almost as high at all other ages.

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STUDIES ON HUMAN LONGEVITY

VI. THE DISTRIBUTION AND CORRELATION OF VARIATION IN THE TOTAL IMMEDIATE ANCESTRAL LONGEVITY OF NONAGENARIANS AND CENTENARIANS, IN RELATION TO THE INHERITANCE FACTOR IN DURATION OF LIFE[1]

BY RAYMOND PEARL AND RUTH DEWITT PEARL

I. THE PROBLEM

FOR the purposes of the following discussion "Total Immediate Ancestral Longevity" (abbreviated TIAL, pronounced *tee-aal*) is taken to mean the sum of the ages at death of the six immediate ancestors of an individual (father, mother, father's father, father's mother, mother's father, mother's mother). Thus if, in the pedigree shown as Figure 1, the letters within the circles of the sex symbols denote ages at death, for the individual (propositus) I,

$$\text{TIAL}_{\text{I}} = a + b + c + d + e + f.$$

TIAL is a definite attribute of biological significance pertaining to the individual (propositus) I, just as truly as his own age is one of his characters. Individuals differ among themselves in respect of their TIAL just as they differ in respect of stature. It may be said of a particular individual A, for example, that he has a stature of 6 ft. 2 in., and a TIAL of 468 years. In one important respect TIAL differs from most other characters of the individual. That is that he shares his TIAL identically with his full siblings. Each of his brothers and sisters necessarily have the same TIAL as he.

TIAL is plainly a character worthy of eugenic consideration and selection. There can be but little argument as to its biological and social

[1] From the Department of Biology of the School of Hygiene and Public Health of the Johns Hopkins University. The authors are indebted to Dr. John R. Miner and Miss Marjorie Gooch for help in various ways in connection with this study.

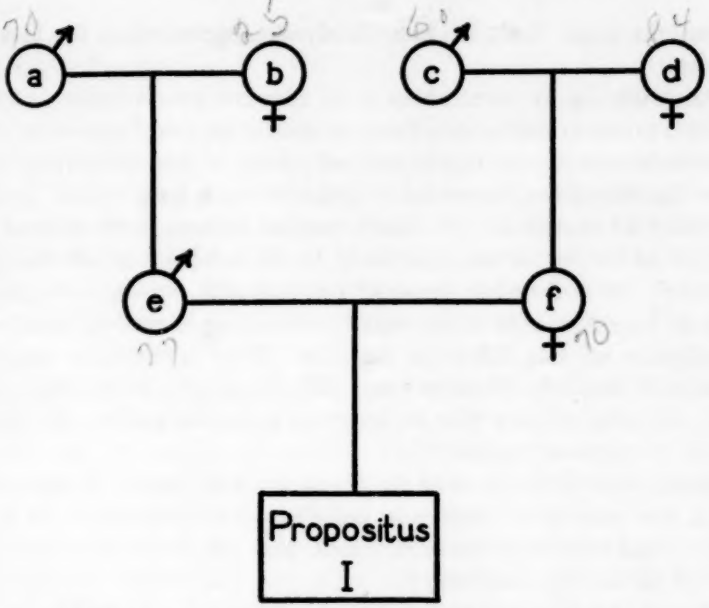

FIG. 1. A PEDIGREE TO DEFINE THE VALUE OF TIAL

worth, of the sort there can be and is about various physical and mental characters. Survival is well-nigh universally counted a biological good. If this were not so preventive medicine and public health would be undesirable efforts. Insofar as longevity is a heritable characteristic TIAL is an attribute of prime importance.

It is evident that the value of TIAL can only vary between upper and lower limits fixed by six times the upper end of the human life span on the one hand, and six times the age of puberty on the other hand. Thus TIAL values are not likely to be found to be much above 600 years on the upper side, and the lower limit will probably not be below 90 years.[2] As a matter of fact neither of these approximate limits has been reached in any actual family in our collection of data. As will presently

[2] For a number of years we have been interested in collecting records to the end of finding the youngest known *living* age at the time of becoming a grandparent. Around 29 years is the lowest figure to come to our attention, and it seems unlikely that any record much lower will be found. Of course the age of *dead* grandparents, with which we are alone concerned here, may be anything from puberty on. We have assumed an age of 15 years for each ancestor in the estimated lower limit of TIAL given above.

appear, the upper limit is more closely approached than the lower in our data.

Sometime ago it occurred to us to examine the variation in TIAL among a group of individuals living at ages of 90 years and over. Such individuals constitute a highly selected group of human beings, in the sense that they have succeeded in living a much longer time than the generality of mankind. No doubt various factors, both internal and external to the individual, contribute to the achievement of this great longevity. It is definitely known [3] that heredity, among other things, plays an important rôle in the matter. But there is need of much more investigation of this factor in the case. It is desirable to study the genetics of longevity from as many different angles as possible. It is from this point of view that we approach the investigation of TIAL in extremely longevous people.

What, then, is the form of the frequency distribution of variation in TIAL for extremely longevous individuals, nonagenarians or centenarians, and how does it compare with that for people in general, not selected for extreme longevity?

This is the first problem of the present study. As will be seen it leads to other matters, particularly those relating to the variation and correlation of various combinations of its component elements.

II. MATERIAL

The data here discussed come from two sets of material in the archives of the Department of Biology. That relating to the extremely longevous individuals living at ages of 90 years and above is derived from our collection of case histories of extreme longevity.[4] This collection has been in process of formation during the past ten years and now includes in total over 1500 finished cases, and about a thousand more still in process of completion. They have been got by the questionnaire method. Originally the questionnaires were sent only to living persons alleged to be 95 years of age and more. As the work has gone on, however, we have been compelled by a sort of social pressure to include some cases between 90 and 95 years of age. The final

[3] Cf. Pearl, R. Studies on human longevity. IV. The inheritance of longevity. Preliminary report. HUMAN BIOLOGY, Vol. 3, pp. 245-269, 1931.

[4] Pearl, R. Preliminary account of an investigation of factors influencing longevity. *Jour. Amer. Med. Assoc.*, Vol. 82, pp. 259-264, 1924.

form of the questionnaire after various revisions is shown in reduced facsimile in Figures 2-5.

These blank forms have, in most cases, been actually filled out by some relative of the aged individual—son, daughter, niece, nephew, or other—in consultation with the living propositus. When the blank is returned the work upon it in the laboratory begins. First every item of the record is gone over critically, and with the attitude that the data are incorrect unless definite evidence of their correctness can be produced. This has two results. The first is that many of the returned blanks are discarded because confirmatory evidence does not develop. The other is that a long correspondence with the individual and his or her relatives is entered upon to bring out information that will, we hope, make the case in the end satisfy our critical standards. If the matter turns out this way, the case is eventually finished and goes into the file for permanent preservation and use. If it does not the case is dropped, and goes into the reject file or the waste basket.

The material includes persons from a great variety of ranks and walks of life. Originally they stem mainly from the racial stocks of northern and central Europe, but mostly have been a long time in this country. The age and sex distribution of the first 1579 cases (from which some may possibly still be rejected because of uncertainty as to the reliability of some of the records involved) is shown in Table 1.

Regarding this Table 1 it should be said that the distributions are in no sense random samples of the number living in the general population at the indicated ages. There has been no attempt to make them such, and the method used in collecting the material makes it impossible for them to be random samples. To get case histories of extremely longevous persons one must first have their names and addresses. Our source for these names and addresses has been primarily newspaper clippings. This means that, in the main, this material contains only extremely old persons whose great age *per se*, or something they did, or that happened to them, caused them to be written about in their local newspapers. This accounts further for the excess frequencies at ages 90, 95, and 100, in contradistinction to the usual reason for such abnormal frequencies at quinquennial ages in census returns. In the data which finally pass our critical tests we know that the persons put down as 90, or 95, or 100 years old, were in actual fact just that old, because we have definite evidence as to date of birth, etc. But why we know about these persons *at all* is because something connected with their

THE JOHNS HOPKINS UNIVERSITY
INSTITUTE FOR BIOLOGICAL RESEARCH

Investigation of Longevity

By filling in the information asked for on this form, you will be greatly aiding the program of our investigation as to the factors which influence longevity. If for any reason you are yourself unable to write in the information desired, will you not please get someone in your household to fill it in for you. This form, after filling out, should be returned in the addressed stamped envelope enclosed herewith to DR. RAYMOND PEARL, Institute for Biological Research, 1901 East Madison Street, Baltimore, Maryland.

NAME

ADDRESS

WHERE WERE YOU BORN? WHEN WERE YOU BORN?

If born abroad in what year did you COME TO THIS COUNTRY?

How OLD were you when you came?

How many BROTHERS did you have? How many SISTERS did you have?

Are any of your brothers and sisters alive now?

If so, give name and address.

How MANY TIMES have you been MARRIED? What was your AGE when MARRIED?

DATE of MARRIAGES?

Give NAME of your first husband-wife.

How old was he-she at death?

When did he-she die (date)?

Give NAME of your second husband-wife.

How old was he-she at death?

When did he-she die (date)?

Was your HUSBAND'S-WIFE'S FAMILY especially LONG-LIVED?
(Give any particulars that you know of.)

How many CHILDREN have you had? BOYS? GIRLS?

If you were married more than once specify how many CHILDREN BY EACH HUSBAND-WIFE

How many of your CHILDREN are NOW LIVING?

How many GRANDCHILDREN have you had?

How many of your GRANDCHILDREN are NOW LIVING?

How many GREAT-GRANDCHILDREN have you had? PLEASE TURN OVER

FIG. 2. FIRST PAGE OF LONGEVITY QUESTIONNAIRE IN REDUCED FACSIMILE

YOUR FAMILY		
RELATIVE	Put in this column the AGE OF THE RELATIVE AT DEATH, OR if LIVING, the AGE NOW.	Put in this column the CAUSE OF DEATH so far as you know it. If person is not dead, write "LIVING."
Your FATHER		
Your MOTHER		
Your FATHER'S FATHER		
Your MOTHER'S FATHER		
Your FATHER'S MOTHER		
Your MOTHER'S MOTHER		
Your CHILDREN (list each child separately by NAME) here	Put in this column the AGE OF EACH CHILD at DEATH, or if LIVING, the AGE NOW.	Put in this column the CAUSE OF THE CHILD'S DEATH, so far as you know it. If child is not dead, write "LIVING."
1st		
2nd		
3rd		
4th		
5th		
6th		
7th		
8th		
9th		
10th		
11th		
12th		
Your BROTHERS and SISTERS (List each one separately) NAME here	Put in this column the AGE OF EACH BROTHER AND SISTER AT DEATH, or if LIVING, the AGE NOW.	Put in this column the CAUSE OF THE BROTHER'S OR SISTER'S DEATH. For those not dead, write "LIVING."

FIG. 3. SECOND PAGE OF LONGEVITY QUESTIONNAIRE IN REDUCED FACSIMILE

PERSONAL HABITS AND HEALTH

To what extent have you USED ALCOHOLIC BEVERAGES during your life?

WINE? BEER?

WHISKEY or other SPIRITS?

To what extent and in what form have you USED TOBACCO?

PIPE? CIGARS?

CIGARETTES? CHEWING?

SNUFF?

How has your HEALTH been generally throughout life?

Have you ever had MEASLES? SCARLET FEVER? WHOOPING COUGH?

TYPHOID FEVER? MALARIA? SMALL POX? PNEUMONIA?

DIPHTHERIA? GOITER? OTHER SERIOUS ILLNESS?

Have you ever undergone a SURGICAL OPERATION?

If so, please state its NATURE, and the DISEASE for which it was undertaken?

WHAT WAS YOUR AGE AT THE TIME?

Please state any other DETAILS ABOUT YOUR HEALTH which you think might be of interest.

What have been your general HABITS during life as to EATING, DRINKING, SLEEPING and WORKING?

TO WHAT DO YOU CHIEFLY ATTRIBUTE YOUR LONG LIFE?

PLEASE TURN OVER

FIG. 4. THIRD PAGE OF LONGEVITY QUESTIONNAIRE IN REDUCED FACSIMILE

RESIDENCE, OCCUPATION, ETC.

In what PLACES have you RESIDED at different times in your life?

Have you LIVED mostly in the COUNTRY or CITY?

What OCCUPATIONS have you followed at different times during life?

To what extent have you done HARD MANUAL LABOR?

What is your RELIGIOUS FAITH?

To what RACE STOCK (English, Scotch, Irish, German, French, etc.), do you chiefly belong?

What is your HEIGHT? AVERAGE WEIGHT?

How has your WEIGHT CHANGED since you were 25 years of age?

What, in general, has been your BUILD DURING ADULT LIFE?

A. THIN AND LEAN?

B. MODERATELY THICK-SET OR CHUNKY?

C. DISTINCTLY FAT?

Color of hair at age 25? Now?

Color of eyes?

Were you a blond or a brunette?

BY WHOM WAS THIS BLANK FILLED OUT?

WHAT IS YOUR RELATION TO

PLEASE GIVE ME THE NAME AND ADDRESS OF ANY OTHER RELATIVE WHO MIGHT BE ABLE TO FURNISH ADDITIONAL OR MISSING INFORMATION

DATE WHEN THIS BLANK WAS FILLED OUT PLEASE TURN OVER

FIG. 5. FOURTH PAGE OF LONGEVITY QUESTIONNAIRE IN REDUCED FACSIMILE

TABLE 1

Family Histories of Extremely Old Persons Living at the Time of Record

AGE OF LIVING PERSON *years*	NUMBER OF CASES		
	Males	Females	Total
90	82	63	145
91	49	35	84
92	62	33	95
93	37	30	67
94	43	37	80
95	77	82	159
96	76	70	146
97	56	53	109
98	45	48	93
99	46	58	104
100	75	141	216
101	31	65	96
102	15	52	67
103	14	22	36
104	11	19	30
105	2	22	24
106	2	6	8
107	4	2	6
108	1	5	6
109	..	2	2
110	..	..	..
111	2	1	3
112	..	2	2
113	..	1	1
Totals	730	849	1579

attainment of these anniversaries was, in each case, given local newspaper publicity. On the other hand, we have no record at all of the relatively large number of persons who got no publicity when they became 90, 95, or 100 years old.

From the finished longevity histories we have extracted for this present study every single case, without any selection whatever, for which the age at death of each of the two parents and four grandparents was recorded. There were 365 such cases. They constitute what will for convenience be called the *Long. Series* in this study. The propositi in this series, it may be repeated for clarity and emphasis, are, in each case, persons *living* at ages of 90 years or over.

Another series of similar data, for purposes of comparison, was extracted from the Family History Records of the Department of Biology. This collection of Family History Records has been used in various investigations [8] and the characteristics of the material described in detail, so that it is not necessary to say anything further about it here. In particular the characteristics of this material in respect of duration of life have been discussed in No. IV of the series of *Studies on Human Longevity*. In the present instance we extracted from the Family History Records every case in which both parents and all four grandparents were dead, and had the ages at death recorded. Since every case in the Records was taken, it is obvious that there could have been no selection *relative to the age of the propositus* to which the TIAL

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[8] Pearl, R. (*a*) The relative influence of the constitutional factor in the etiology of tuberculosis. *Amer. Rev. Tuberc.*, Vol. 4, pp. 688-712, 1920.

———. (*b*) New data on the influence of alcohol on the expectation of life in man. *Amer. Jour. Hyg.*, Vol. 2, pp. 463-466, 1922.

———. (*c*) The age at death of the parents of the tuberculous and the cancerous. *Ibid.*, Vol. 3, pp. 71-89, 1923.

———. (*d*) Alcohol and Mortality. In *The Action of Alcohol on Man*, by E. H. Starling. *London* (Longmans, Green and Co.), 1923.

———. (*e*) Alcohol and life duration. *Brit. Med. Jour.*, Vol. I for 1924, pp. 948-950.

Kacprzak, M. (*f*) Tuberculosis and fertility. *Amer. Jour. Hyg.*, Vol. 4, pp. 605-638, 1924.

Pearl, R. (*g*) Alcohol and Longevity. *New York* (Knopf), 1926.

———. (*h*) Alcool e longevità. *Assicurazione Sociali*, Anno II, No. 5, pp. 22-49, 1926.

———. (*i*) The constitutional factor in breakdown of the respiratory system. *Ann. Eug.*, Vol. 2, pp. 1-24, 1927.

———. (*j*) Alcohol and life duration. *Internat. Clinics*, 38th series, Vol. 3, pp. 28-52, 1928.

———. (*k*) Alkohol und Lebensdauer. *Hannover* (Norddeutsches Druck-und Verlagshaus), 1930.

———. (*l*) Studies on human longevity. IV. The inheritance of longevity. Preliminary report. HUMAN BIOLOGY, Vol. 3, pp. 245-269, 1931.

figure relates, except insofar as the restriction that all six immediate ancestors must be dead may imply such a selection. As a matter of fact, having taken every case in which all six immediate ancestors were dead (*i.e.,* having made the basis of selection of cases an attribute of the *ancestry*) we were compelled to make an arbitrary choice of an individual from the offspring sibship, to play the rôle of propositus (I in Fig. 1) in this series. The rule we decided upon was to take as propositus the oldest *living* individual in the sibship, because the propositus in each case in the Long. Series was living. In 7 cases there was no living sibling. In these cases we took as propositus the individual in the sibship who had the highest age at death.

So, then, we have for comparison in this study two series of data, in one of which the propositus in every case had achieved extreme longevity (living age 90+ years), and in the other of which the propositus in each case was not directly selected in respect of age. The latter series will be designated in this study as the FHR Series. It contains 143 individuals.

Summarily stated, we are comparing in this study two groups of living persons (the propositi). These two groups are exactly alike in one respect, namely that all six immediate ancestors of the individuals in each group are dead. In one of these groups (here called the Longevous Series) each one of the offspring sibships stemming from the six dead ancestors contains at least one person 90 years of age or over and still living, and for the propositi in this group one such nonagenarian or centenarian is chosen for each offspring sibship. In the other group (here called the FHR Series) no attention whatever is paid to the ages of the persons in the offspring sibships (but in fact no one of them happens to contain a nonagenarian or centenarian) deriving from their six dead ancestors, and for the propositi in this group merely the oldest living individual in each sibship is chosen (except in the seven cases where there was no living sib).

It should be clearly understood, and is here emphasized in order that there may be no misapprehension about the matter, that the FHR Series is a group of individuals selected for *comparison* with the Long. Series, and nothing but that. It is *neither* a control group in the sense that the word "control" is used in physiological experimentation for example, *nor* a random sample of any general population in any general or inclusive sense of the word "random." In the nature of the case it is *impossible* now or at any other time to obtain a group of persons who

will be, in any strict or inclusive sense of the words, a random control against a series of living nonagenarians and centenarians for the purpose of studying the inheritance of longevity. The best that can be done in the premises is to *compare* the longevous group with a selected sample of persons, the sample being so selected as to lead to as great a degree as possible of pertinent significance in the comparisons made. This has been the motive in forming the FHR Series in the manner described above. At the same time sound scientific methodology demands that the manner of selection of the comparison group, and the statistical consequences automatically and necessarily entailed by the manner of selection, shall be defined and described with as much detail and candor as possible, so that the reader may at every stage of the report form for himself an intelligent judgment as to the meaning and significance of the comparative results set forth. To such definition and description of the FHR Series (and also of the Long. Series) we now address ourselves. Owing to limitations of space we are unable to present here all the pertinent descriptive data relating to some of the more remote consequences of the manner of selection of the FHR Series, but in the expanded publication of this study in book form, the additional material referred to is presented as an Appendix.

We return now to the description of the material.

All of the persons in both Long. and FHR series are *whites*.

The *sex distribution* of the persons in the two series of material is shown in Table 2.

TABLE 2

Sex Distribution of Propositi

	MALES	FEMALES	TOTALS
Long. Series	190	175	365
FHR Series	72	71	143

It is seen that the propositi in both series represent each sex about equally. In most of the computations and discussions both sexes will be dealt with together. The numbers are not large enough for profitable discussion separately.

The frequency distributions and biometric constants relative to the *living age* of the propositi in the two series are shown in Table 3.

TABLE 3

Frequency Distributions and Biometric Constants for Age (Living) of Propositi

AGE IN YEARS	LONG. SERIES	FHR SERIES [6]
10- 19	...	4
20- 29	...	12
30- 39	...	19
40- 49	...	32
50- 59	...	40
60- 69	...	22
70- 79	...	6
80- 89	...	1
90- 94	173	...
95- 99	133	...
100-104	52	...
105-109	7	...
Totals	365	136
Mean	95.43 ± .13 yrs.	48.75 ± .83 yrs.
Median	94.95 ± .17 yrs.	50.25 ± 1.04 yrs.
Standard deviation	3.79 ± .10 yrs.	14.40 ± .59 yrs.
Coefficient of variation	3.97 ± .10%	29.53 ± 1.31%

It is evident from the figures in Table 3 that the individuals composing the Long. Series are a highly selected lot, showing little variation, either absolute (standard deviation) or relative (coefficient of variation) in respect of age. Their mean (and median) ages testify in another way to the rigor of the selection represented, since only about four persons in a thousand born alive ever reach the age of 95 years.

The individuals in the FHR Series, on the other hand, are seen to furnish a group that appears to be from certain viewpoints profitably usable for comparison with the longevous. Life tables [7] give the expectation of life (mean after-lifetime) at birth in 1901 (which appears to

[6] With the seven dead propositi omitted, leaving 136 as the number in the distribution from which these constants are computed. The constants for the Long. Series were computed from a distribution with two year class units.

[7] Glover, J. W. United States Life Tables 1890, 1901, 1910, and 1901-1910. *Washington* (Bureau of the Census), 1921.

be the date most nearly comparable for the present data) for white persons in cities of the Original Registration States, as 47.32 years for males, and 47.90 years for females. These values are not widely different from the mean living age of the propositi in our FHR Series. That series may therefore be taken as a group of persons who had lived, on the average, about the same length of time that people in general live, on the average, after birth.

It has been pointed out earlier that there was no selection of the propositi in the FHR Series relative to their own living age except such as may have been involved (a) in taking as propositus the oldest living individual in each sibship, and (b) in requiring that all six immediate ancestors should be dead, and their ages at death known. Furthermore, it has just been shown that the mean living age of the propositi in this series is not greatly different from the expectation of life at birth set down in a fairly comparable U. S. life table. But so simple and rough a test as this is not sufficient. It is necessary before we go on to use the FHR Series to compare with the Long. Series to learn more precisely the effects of procedures (a) and (b) *supra.* To do this it is necessary to get the answers to at least four specific questions, as follows:

1. What are the values of the biometric constants relative to age of the total living population at the 1930 Census?

2. What are the values of the biometric constants relative to age of all the living persons (including the propositi themselves) in the 136 sibships from which the propositi in the FHR Series were drawn as oldest living individuals?

3. How do the frequency distributions of ages of (α) the propositi in the FHR Series, and of (β) all the living individuals in the sibships from which the propositi were drawn as oldest individuals compare with the similar distribution of (γ) the total living white population at the 1930 Census?

With the answers to these three questions in hand it will be possible to form a reasonable judgment as to the effect upon the FHR Series of procedure (a) *supra,* that is the taking of the oldest living individual as propositus.

It will be necessary then to consider:

4. What will be the frequency distribution and biometric constants of a series of say 200 individuals, each the oldest living in the sibship to which he or she belongs, these sibships being taken at random, and

without reference to whether the six immediate ancestors are living or dead, except for the requirements that the ages of all of them shall be recorded and that at least one of them shall be living?

The answer to this question will permit some judgment to be formed as to the effect of procedure (b) *supra*, that is the requirement in making the FHR Series that all six immediate ancestors must be dead.

In answer to questions 1 and 2 Table 4 presents the age data for the living population of the United States.[8]

TABLE 4

Biometric Constants for Age (Living) of (γ) White Population of United States (1930 Census. Both Sexes); and (β) Total Living Persons in Sibships From Which FHR Series Propositi Were Drawn

	γ	β
	U. S.	FHR Series Total Sibships (living)
Mean	29.864± .001 yrs.	45.774± .407 yrs.
Median	26.975± .002 yrs.	45.638± .510 yrs.
Standard deviation	19.942± .001 yrs.	13.704± .288 yrs.
Coefficient of variation	66.776± .004 %	29.94 ± .68 %
N	108,786,878	517

From Table 4 we note:

The living members of the sibships from which the propositi of the FHR Series were drawn plainly form a selected group differentiated in respect of age from the total living white general population. This selection (and differentiation) is in the same direction as that characterizing the Long. Series, but is less intense (that is, has not proceeded so far in the same direction) than in the Long. Series. The total living sibships have a mean age nearly 16 years higher than that of the living whites of the general population. The median age is over 18 years higher in the total sibling group. The variability of the total

[8] Fifteenth Census of the United States: 1930. Population, Vol. III, Part 1. *Washington* (Gov't Printing Office), 1932. Data from p. 14.

living sibships to which the FHR propositi belong, whether measured absolutely (S. D.) or relatively (C. of V.) is significantly less than that of the living white general population. Altogether it appears that, in comparing our Long. Series with the FHR Series, we are making a much less drastic comparison, so far as concerns the age of propositus, than would be the case if we were able to compare the Long. Series with the living general population.

The relations of the basic constants of the four series of data under discussion are shown graphically in Figure 6.

In the order of arrangement as depicted in Figure 6 the mean ages of the four series stand in the ratios 1 : 1.63 : 1.53 : 3.20, and the standard deviations in ages in the ratios 1 : 0.72 : 0.69 : 0.13.

2. The distribution of variation in the four series under discussion may well be exhibited at this point in the discussion. They are shown graphically in Figure 7, on a relative basis, the frequencies being plotted as percentages for 10 year intervals of age.

This Figure 7 shows in graphic form the differences between the several series that have already been deduced from the biometric constants. The greater skewness and variability of the general population is striking. The extreme differentiation of the propositi of the Long. Series from all the others is also made abundantly clear in this diagram.

3. If the final columns of Tables 3 and 4 are compared it is plain that the selection of the oldest living individual in the sibships as propositus in the FHR Series made no serious difference from what the result would have been if all living sibs had been taken. The mean age of the FHR propositi is $2.98 \pm .93$ years higher than that of the total living sibships from which they were drawn. While this difference is slightly more than three times its probable error, and therefore probably significant, it is absolutely so small as to make no practical difference in any conclusions likely to be drawn in such a study as the present one. In variability, as measured by the standard deviations, the value for the FHR propositi is $0.70 \pm .66$ years greater than that for the total living sibships from which they were drawn. This is an obviously insignificant difference, as is also that between the coefficients of variation. But whether the oldest living individual in the sibship be taken or all the living sibs, the FHR Series is markedly differentiated from the general population of living persons. This appears to be primarily a resultant of the fact that the FHR propositi come from *completed* families, in the sense that their parents (being dead) are not going to

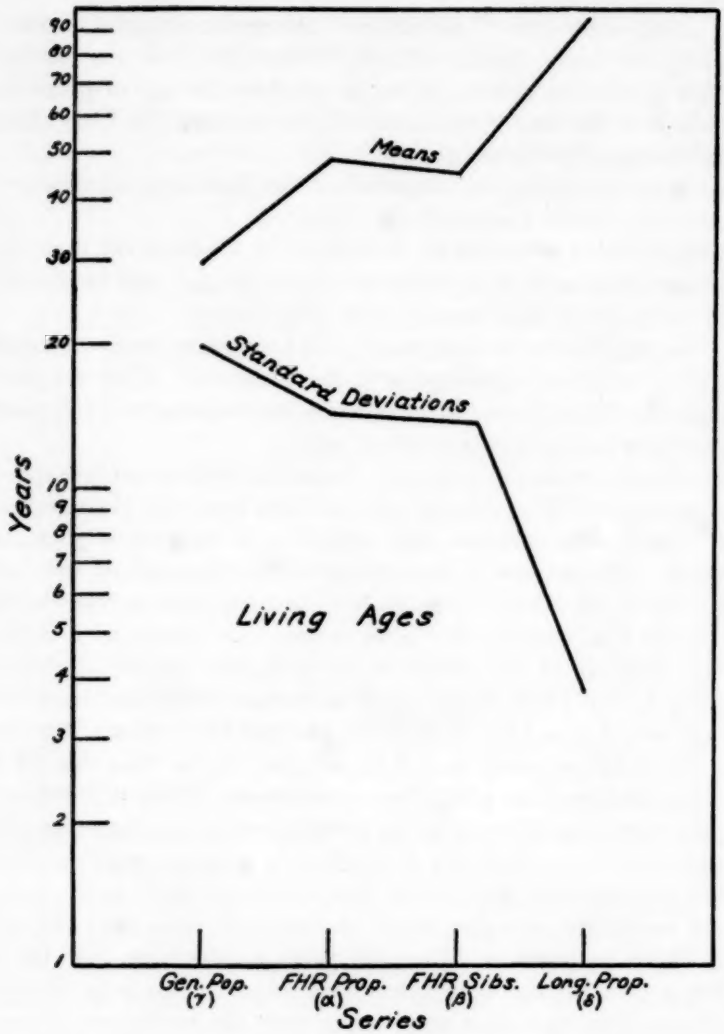

FIG. 6. VALUES OF THE MEANS AND STANDARD DEVIATIONS FOR AGE OF (α) PROPOSITI IN FHR SERIES; (β) ALL LIVING INDIVIDUALS IN SIBSHIPS FROM WHICH PROPOSITI WERE DRAWN; (γ) TOTAL LIVING WHITE POPULATION OF U. S.; (δ) PROPOSITI IN LONG. SERIES

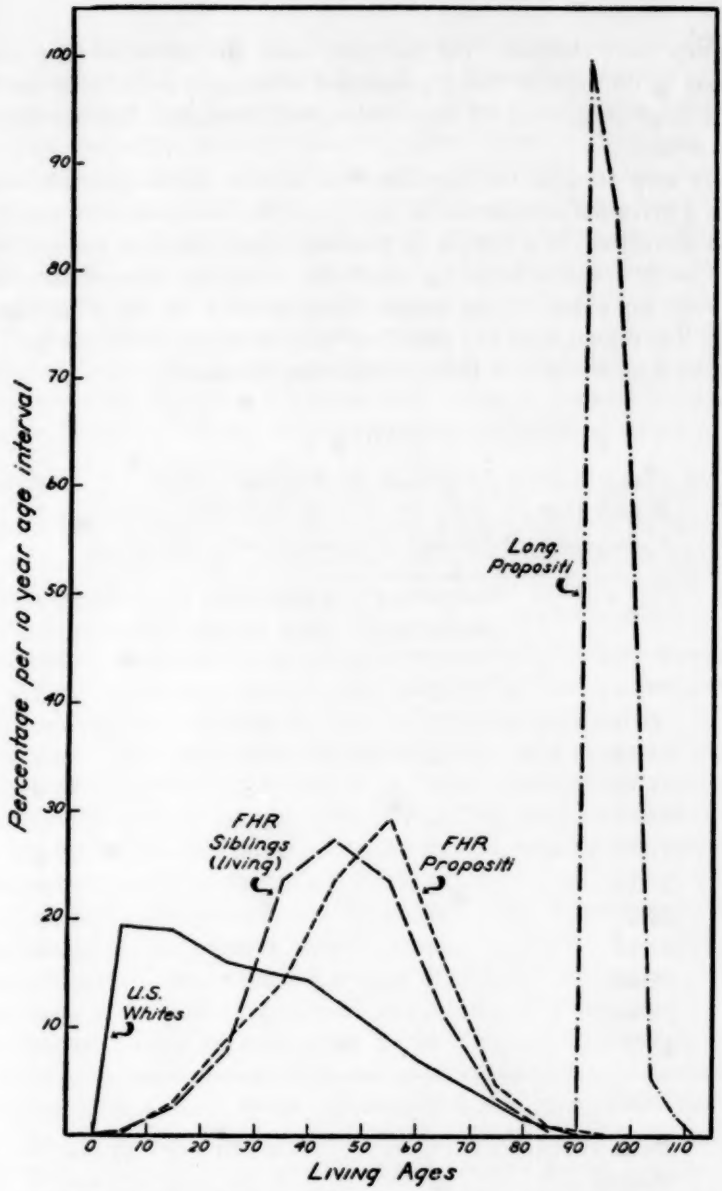

FIG. 7. FREQUENCY POLYGONS OF VARIATION IN AGE OF (α) PROPOSITI IN FHR SERIES (SHORT DASH LINE); (β) ALL LIVING INDIVIDUALS IN SIBSHIPS FROM WHICH PROPOSITI WERE DRAWN (LONG DASH LINE); (γ) TOTAL LIVING WHITE POPULATION OF U. S., 1930 (SOLID LINE); (δ) PROPOSITI IN LONG. SERIES (DOT-DASH LINE)

have any more children. On the other hand, the excess of very young persons in the general living population arises primarily from the fact that a large proportion of them belong to incomplete families (in the same sense).

We now turn to the consideration of our fourth question *supra*. Table 5 gives the distribution of ages, and the biometric constants computed therefrom, in a sample of 200 individuals taken at random from our Family History Records, under the following rules of sampling: (a) each individual is the oldest living member of the sibship from which it is drawn, and (b) one (any one) or more of his (or her) six immediate ancestors was living at the time of record.

TABLE 5

Age of Oldest Living Individual in Random Sample of Sibships in Which One or More of His (or Her) Six Immediate Ancestors (2 Parents, 4 Grandparents) was Living

AGE	FREQUENCY (ABSOLUTE)	FREQUENCY (PER CENT)	ACCUMULATED PERCENTAGES
0- 4	5	2.5	2.5
5- 9	11	5.5	8.0
10-14	13	6.5	14.5
15-19	17	8.5	23.0
20-24	34	17.0	40.0
25-29	37	18.5	58.5
30-34	25	12.5	71.0
35-39	28	14.0	85.0
40-44	14	7.0	92.0
45-49	8	4.0	96.0
50-54	6	3.0	99.0
55-59	2	1.0	100.0
Totals	200	100.0	....

Mean	$= 28.03 \pm .57$ yrs.
Median	$= 27.70 \pm .71$ yrs.
Standard deviation	$= 11.89 \pm .40$ yrs.
Coefficient of variation	$= 42.4 \pm 1.7$ %

It is at once apparent that the requirement set up for the drawing of our sample of FHR propositi that all six immediate ancestors should be dead—that is that the propositi should belong to completed families—has a marked effect in determining the distribution of their ages. The mean age of the FHR propositi is 20.72 ± 1.01 years higher than that of the individuals of Table 5, and the median age is 22.55 ± 1.26 years higher. The individuals of Table 5 are somewhat less variable absolutely ($\text{Diff.}_{\text{S.D.}} = 2.51 \pm .71$ years), but significantly more variable relatively ($\text{Diff.}_{\text{C. of V.}} = 12.9 \pm 2.2$ per cent) than the FHR propositi.

Taking families not necessarily completed, as is done in Table 5, considerably curtails the age distribution of the oldest living individuals in the offspring sibships at the upper end. None of the individuals is 60 or more years old. Forty per cent of them are under 25 years of age, and nearly 60 per cent are under 30 years.

The close similarity of the constants of Table 5 with those of the first column of Table 4 (the total living white population of the U. S., 1930) is most striking. The means differ by only 1.83 ± .57 years; and the medians by only 0.73 ± .71 years, the Table 5 sample being higher in the one case and lower in the other. The Table 5 sample is significantly less variable, as might be expected, both from the size of the sample, and much more from the fact that in Table 5 the method of taking the sample excludes all *necessarily* completed families. But the similarity of the centering constants brings out once more the essential normality of our Family History Records in respect of age distribution as compared with the general population. This has been noted before,[8] but has sometimes been overlooked, denied, or even misrepresented by critics of our work on alcohol and life duration.

There is, finally, another way in which the age characteristics of the two groups studied may be stated. We are comparing two groups of living persons. One of these groups (FHR Series) has an average living age at the time of record of 48.75 years, and consequently from Glover's [7] life table for both sexes in the Original Registration States as of 1901 has an expectation of life of 22.13 years. It is therefore to be expected that when all the individuals in this group have died their average age at death will turn out to be 70.88 years or thereabouts. The other group of living persons (Long. Series) has an average living age at the time of record of 95.43 years, and an expectation of life of

[8] *Cf.* Pearl, R. Alcohol and Longevity, *passim.*

TABLE 6

Birthplace and Habits Relative to Alcohol of Persons in the Long. Series (Nonagenarians and Centenarians)

PLACE OF BIRTH	ABSTAINERS		VERY MODERATE		MODERATE		HEAVY		VERY HEAVY		TOTALS			
Native Born	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	Both sexes	Per cent
New York	14	24	3	3	7	1	2	..	..	..	26	28	54	14.9
Massachusetts	8	24	8	5	8	..	..	..	..	..	24	29	53	14.6
Pennsylvania	8	10	5	5	3	1	..	..	..	..	16	16	32	8.8
Ohio	9	11	3	3	4	1	..	..	..	..	16	15	31	8.6
Maine	5	7	4	4	2	1	..	..	..	..	11	12	23	6.4
Connecticut	6	5	4	2	1	..	..	..	..	..	11	7	18	5.0
Virginia	2	7	1	1	3	1	..	..	..	..	6	9	15	4.1
New Hampshire	4	2	1	6	1	..	..	..	..	..	6	8	14	3.9
Vermont	3	6	1	1	1	..	..	..	..	..	5	7	12	3.3
New Jersey	4	3	1	2	1	..	..	..	..	..	6	5	11	3.0
North Carolina	1	4	2	..	1	1	..	..	1	..	5	5	10	2.8
Indiana	6	2	..	..	..	2	..	..	..	..	6	4	10	2.8
Maryland	..	2	2	..	2	1	..	..	..	..	4	3	7	1.9
South Carolina	2	1	..	..	..	1	1	..	..	..	3	2	5	1.4
Illinois	1	..	3	1	..	..	..	..	..	..	4	1	5	1.4
Georgia	..	..	2	..	2	..	..	..	..	..	4	..	4	1.1
Rhode Island	1	..	..	1	1	..	..	..	..	..	2	1	3	0.8
Kentucky	..	..	2	..	1	..	..	..	..	..	3	..	3	.8
Tennessee	1	..	..	..	..	..	..	..	1	..	2	..	2	.6
Michigan	1	..	..	..	1	..	..	..	..	..	2	..	2	.6
Delaware	..	..	..	1	1	..	..	..	..	..	1	1	2	.6
Alabama	..	..	1	..	..	..	..	..	..	..	1	..	1	.3
Missouri	1	..	..	..	..	..	..	..	..	..	1	..	1	.3
Florida	1	..	..	..	..	..	..	..	..	..	1	..	1	.3

Missouri	1	..	..	..	..	..	..	..	..	..	1	..	1	.3
Florida	1	..	..	..	..	..	..	..	..	..	1	..	1	.3

TABLE 6—*Continued*

PLACE OF BIRTH	ABSTAINERS		VERY MODERATE		MODERATE		HEAVY		VERY HEAVY		TOTALS			
Foreign Born	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	Both sexes	Per cent
Ireland	3	2	..	1	1	..	..	..	..	..	4	3	7	1.9
Germany	1	..	..	..	4	1	..	..	..	..	5	1	6	1.7
England	..	2	1	2	1	..	..	..	..	..	2	4	6	1.7
Norway	1	2	..	1	1	..	..	..	..	..	2	3	5	1.4
Canada	..	1	..	..	2	..	1	..	1	..	4	1	5	1.4
Scotland	1	1	..	1	..	..	..	..	..	..	1	2	3	0.8
Holland	..	2	..	..	1	..	..	..	..	..	1	2	3	.8
Nova Scotia	..	1	1	..	..	..	..	..	..	..	1	1	2	.6
Wales	..	1	..	..	..	..	..	..	..	..	..	1	1	.3
Russia	1	..	..	..	..	..	..	..	..	..	1	..	1	.3
Italy	..	..	..	..	1	..	..	..	..	..	1	..	1	.3
Finland	..	..	1	..	..	..	..	..	..	..	1	..	1	.3
Belgium	..	..	..	..	..	1	..	..	..	..	..	1	1	.3
Panama	..	..	1	..	..	..	..	..	..	..	1	..	1	.3
Totals	85	120	47	40	51	12	4	..	3	..	190	172	362*	100.4
Percentages by sex as to alcohol	44.7	69.8	24.7	23.3	26.8	7.0	2.1	..	1.6	..	99.9	100.1		
Group totals	205		87		63		4		3		362			
Group percentages	56.6		24.0		17.4		1.1		0.8		99.9			

* Three cases are missing because there was no information given regarding the use of alcohol. These are Cases Nos. 607, 1630, and 2200.

2.12 years, approximately one-tenth of that of the other group. When this second group is all dead the average age at death of all the individuals in the group will turn out to be about 97.55 years.

To summarize this long discussion of the age characteristics of the propositi in the material of this study, it may be said that:

1. Detailed data have been presented which enable the reader to understand precisely the nature, in respect of living age of the propositi, of the two sets of data (Long. Series and FHR Series) that we propose to discuss.

2. Taking the oldest living individual in the sibship as propositus in the FHR Series gives no essentially different result, for practical purposes, than if all living individuals in the sibship had been taken together as a sort of composite propositus. Furthermore there is an element of fairness in taking the oldest living individual for comparative purposes because in the Long. Series the propositus is always the oldest living member of the sibship to which he (or she) belonged.

3. Because the FHR propositi come from necessarily completed families their mean age is higher and their variability in age is lower than is the case in a random sample of all living individuals in the general population. This means that in using them for comparison with the Long. Series we are making a less severe contrast than would be the case if incomplete families were used. But the comparisons actually made in this paper would, on just that account, appear to be all the fairer, because the individuals in the Long. Series come from necessarily completed families (both parents being long since dead).

Table 6 presents data regarding the birthplace (from which some judgment as to race stock may be formed) and habits relative to the use of alcohol by the persons in the Long. Series (nonagenarians and centenarians). Data on these points for the material from which the FHR Series is drawn have already been given elsewhere [10] and need not be repeated here.

The classification of the individuals in Table 6 relative to their usage of alcohol as a beverage is based upon the *maximum* used at any period of their lives. This seems as reasonable a basis as any for a general exhibition of the characteristics of the material. While the drinking habits of individuals may change in the course of such long

[10] Pearl, R. Alcohol and Longevity, *passim.*

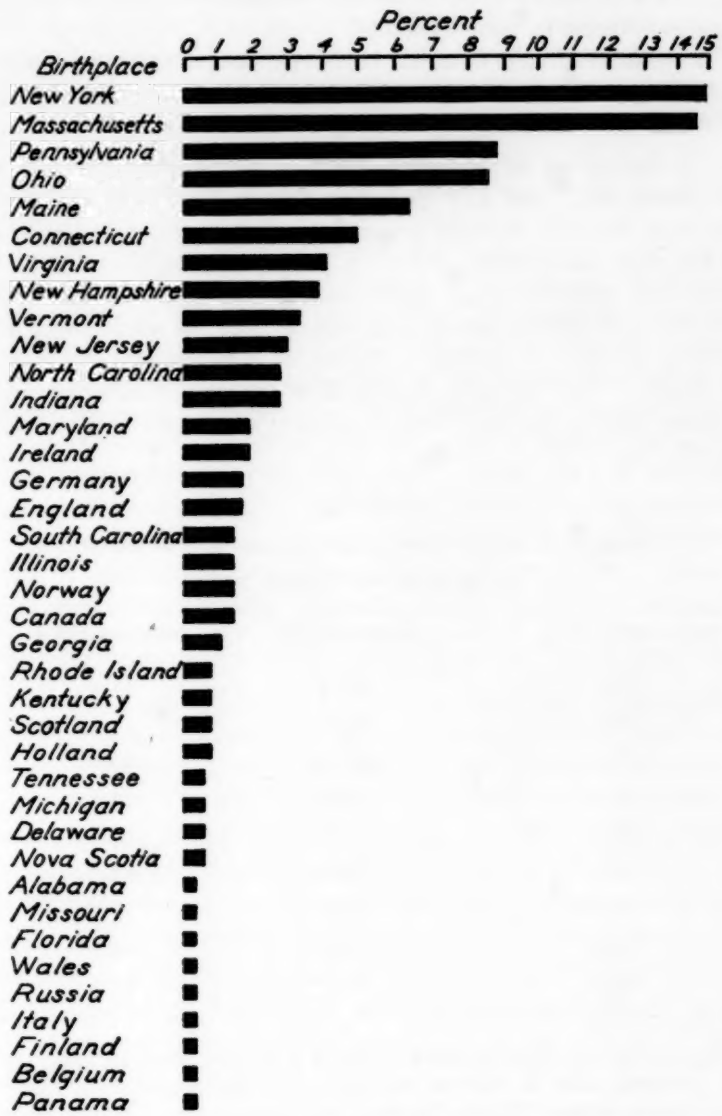

FIG. 8. BAR DIAGRAM SHOWING THE BIRTHPLACE OF 362 LIVING NONAGENARIANS AND CENTENARIANS. THE LENGTHS OF THE BARS ARE PROPORTIONAL TO THE NUMBER BORN IN EACH PLACE.

lives, it will be useful to see what was the maximum of potatory performance achieved by each individual.

The data of Table 6 as to birthplace are shown graphically in Figure 8.

From Table 6 and Figure 8 the following points may be noted:

1. In this group of nonagenarians and centenarians 88.3 per cent were native-born, and 11.7 per cent foreign-born. In the United States as a whole in 1930 the proportions of the white population were 87.7 per cent native-born, and 12.3 per cent foreign-born. It thus appears that these living nonagenarians and centenarians have grossly about the same distribution as to nativity as does the whole living white population at the present time. But let us examine into the matter a little further. The following figures show the approximate percentages of foreign-born in the total white population at the census enumerations from 1850 on.

TABLE 7

Percentages of Native-born and Foreign-born in the White Population of the United States

CENSUS	NATIVE-BORN	FOREIGN-BORN
1850 [11]	88.5	11.5
1860	84.6	15.4
1870	83.4	16.6
1880	84.6	15.4
1890	83.2	16.8
1900	84.5	15.5
1910 [12]	83.7	16.3
1920 [13]	85.9	14.1
1930	87.7	12.3

[11] Figures for 1850-1900 inclusive computed from data given in Tables 17 and 54 of reference cited in footnote 16 *infra*. The percentages given reckon all foreign-born as whites. This is not strictly true, but the error is insignificant.

[12] Computed from data given in Table 17 of the reference cited in footnote 8 *supra*.

[13] Figures for 1920 and 1930 from Table 3 of the reference cited in footnote 8 *supra*.

It is evident that the close approach of the foreign-born percentages of the Long. Series is somewhat fortuitous. If immigration restrictions had not operated in recent years to reduce the percentages of foreign-born in the population it would not have been so close as it is. But in 1850, roughly about the time when most of the foreign-born in the Long. Series came to this country, the proportion of foreign-born in the white population was almost exactly the same as that shown by the Long. Series.

2. Just under 30 per cent of the persons in the Long. Series were born in one or the other of the two states, New York and Massachusetts. Fourteen states (Maine, New Hampshire, Massachusetts, Rhode Island, Connecticut, New York, New Jersey, Delaware, Maryland, Virginia, North Carolina, South Carolina, Georgia, and Florida) bordering on the Atlantic seaboard furnished (as birthplace) 60.8 per cent of the total. If Pennsylvania be added to this group, as a seaboard state for all practical purposes, the percentage rises to 69.6. If the reckoning is based upon the native-born portion of the whole group it appears that 68.8 per cent of the sample of nonagenarians and centenarians was born in one or another of the fourteen states lying along the Atlantic seaboard, in the strict sense of the term. If Pennsylvania be included the percentage becomes 78.8. Only one person in the group was born west of the Mississippi River. Only 14.2 per cent of the whole group (native and foreign together) was born south of the Mason and Dixon line. Altogether it is plain that this group of extremely longevous persons now (at the time of record) living originated preponderantly from the northeastern part of the country. If we take as the northeastern part of the country the nine states east of Ohio and north of the Mason and Dixon line it appears that 60.7 per cent of our whole longevous group, and 68.8 per cent of the native-born portion of it, were born in that region. Now these persons were born around a hundred years ago. In 1830 these nine northeastern states contained 51.4 per cent of the total white population of the country. So it is seen that this portion of the country is even more heavily represented in the Long. Series (as birthplace) than its relative population would warrant. This is in part due to the fact that, because of the better early vital records of that part of the country, it has been easier for cases originating there to satisfy the critical requirements for admission into our collection of longevity material.

3. Turning now to the foreign-born portion of this group of nonagenarians and centenarians it comes as something of a surprise to find that the Irish born constitute the largest single national group as to birthplace. The Irish are known to be a rather short-lived people, taken as a group.[14] Germany and England each contribute (as to birthplace) practically the same number as Ireland to this sample of the longevous. Each of these countries has a larger representation than either South Carolina, Illinois, Georgia, Rhode Island, Kentucky, Tennessee, Michigan, Delaware, Alabama, Missouri or Florida. In the total white population of the country in 1930, 0.85 per cent were born in Ireland, 1.48 per cent in Germany, and 0.74 per cent in England.[15] From these figures it appears that while nonagenarians and centenarians of German birth in this material are present in not far from the same proportion as Germans in the 1930 population, the Irish and English are overrepresented in this sample of longevous persons, as compared with the general population of the present time. Unfortunately data are not available as to the country of birth of the foreign-born in 1830. Such information was first included in the census returns of 1850.[16] But in 1850, when the persons of the Long. Series were, generally speaking, in their 'teens, the proportions of persons of English, Irish, and German birth in the population were as follows:

TABLE 8

Population in 1850

COUNTRY OF BIRTH	NUMBER	PER CENT OF TOTAL WHITES
Ireland	961,719	4.92
Germany	583,774	2.99
England	278,675	1.43

In 1850 there were absolutely more Irish-born persons in the population of the United States—38,077 more to be exact—than there were in 1930, although the total white population was 5.6 times greater in

[14] Dublin, L. I., and G. W. Baker. The mortality of race stocks in Pennsylvania and New York. *Quart. Publ. Amer. Stat. Assoc.*, March, 1920.

[15] See footnote 8 *supra* for source of data. Pp. 7 and 11.

[16] Rossiter, W. S. A Century of Population Growth from the First Census of the United States to the Twelfth 1790-1900. *Washington* (Gov't Printing Office), 1909. Pp. x + 303.

1930 than it was in 1850. So it is perhaps not remarkable that there are more Irish than any other foreign-born in the Long. Series—there were many more Irish in the country when they started life than there were of foreign-born of any other single nationality.

4. Taking the group as a whole, males and females together, a few more than a half (56.6 per cent) of these nonagenarians and centenarians have been total abstainers from alcohol throughout their long lives. This, however, is not true of the males taken by themselves, where but 44.7 per cent fall in the total abstainer class. Among the women nearly 70 per cent (69.8) are abstainers. At the other extreme 3.7 per cent of the males have been heavy or very heavy drinkers at some time in their lives. More than a quarter of the men have been moderate drinkers, mostly throughout their lives. Seven per cent of the women also fall in this class. Altogether these data furnish cogent evidence that the moderate use of alcoholic beverages does not prevent the attainment of great longevity to the user. They further show that some individuals may indulge heavily in alcohol and still become nonagenarians. These observations are incompatible with the widely preached doctrine that any indulgence in alcohol, however slight, inevitably and universally shortens life.

5. The proportion of abstainers in the Long. Series is somewhat larger than that in a sample of the population of Baltimore, previously studied.[17] That sample gave, taking males and females together, a total abstainer percentage of 45.3. The difference between these two percentages (56.6 — 45.3) is 11.3. Is this difference greater than might reasonably appear solely as the result of fluctuations of random sampling? If the two groups are thrown together the percentage of abstainers is 46.0. If this were the true proportion of abstainers in the general population the probable error for the difference between percentages in such samples as the actual ones would be

$$.67449\sqrt{46\times 54}\sqrt{\frac{1}{5248}+\frac{1}{362}}=1.83$$

We must therefore conclude that the 11.6 per cent more abstainers in the Long. Series represents a significant difference.

[17] See footnote 10 *supra*.

6. The "moderate" and "very moderate" classes of the Long. Series together include 41.4 per cent of the total. This compares with the 37.3 per cent of moderate drinkers in the Family History Records sample cited above, since in the former study "moderates" included the two classes here separated. The difference here is 4.1 ± 1.78 per cent, the probable error of the difference being computed as before. This difference cannot be regarded as significant, being only a little more than twice its probable error. So then we conclude that while this group of nonagenarians and centenarians included a higher proportion—slightly over 4 per cent higher—of moderate drinkers than a sample of the general population unselected and undifferentiated in respect of longevity from the average run of the American population, this higher proportion is not greater than might arise from fluctuations of sampling. The odds against its having so arisen are over 7 to 1, but such odds do not constitute certainty.

7. The 3.7 per cent of "heavy" and "very heavy" drinkers amongst the males in the Long. Series compares with 27.4 per cent in the sample of the general population. Here the difference is large and obviously significant.

8. Summarizing the whole situation relative to drinking habits we see that this group of nonagenarians and centenarians includes proportionately somewhat more abstainers and more moderate drinkers than a random sample of the general population of average longevity. It therefore necessarily has fewer heavy drinkers. This result confirms, from an independent source, the general conclusion reached in our earlier study of the influence of alcohol upon longevity, to the effect that while moderate drinking could not be shown to affect adversely the duration of life, heavy drinking was statistically associated with curtailed average longevity. But in that study, just as in this, some heavy drinkers achieved longevity far above the average.

III. RESULTS

A. The Distribution of Variation in TIAL

The frequency distributions, both absolute and relative, of TIAL in the two series are given in Table 9.

From Table 9 several points of interest are immediately evident. In the first place it is plain that the natural range of variation in TIAL is great. Taking the extreme limits of the age classes it amounts to 359

TABLE 9

TIAL Distributions

TIAL (*in years*)	LONG. SERIES		FHR SERIES	
	Absolute	Percentage	Absolute	Percentage
250-269	1	.27	..	...
270-289	..	...	3	2.10
290-309	1	.27	6	4.20
310-329	4	1.10	8	5.59
330-349	5	1.37	14	9.79
350-369	11	3.01	25	17.48
370-389	19	5.21	25	17.48
390-409	34	9.32	15	10.49
410-429	49	13.42	18	12.59
430-449	50	13.70	16	11.19
450-469	81	22.19	10	6.99
470-489	53	14.52	2	1.40
490-509	32	8.77	1	.70
510-529	19	5.21	..	...
530-549	3	.82	..	...
550-569	1	.27	..	...
570-589	1	.27	..	...
590-609	1	.27	..	...
Totals	365	99.99	143	100.00

years (609 — 250). Actually the lowest observed figure was 254 years, and the highest 599 years. Thus while the lowest observed figure is still more than 150 years greater than the physiological lower limit, the highest observed TIAL falls only one year short of the 600 years which measures the situation where each and every one of a person's six immediate ancestors live to be 100 years old. The pedigrees of the two individuals having the extreme values of TIAL are shown in Figures 9 and 10.

The range of variation in TIAL is *greater,* by about 120 years, in the Long. Series than in the FHR Series, although, as will presently appear and would be expected theoretically, the variation as measured by standard deviation and coefficient of variation is somewhat greater in

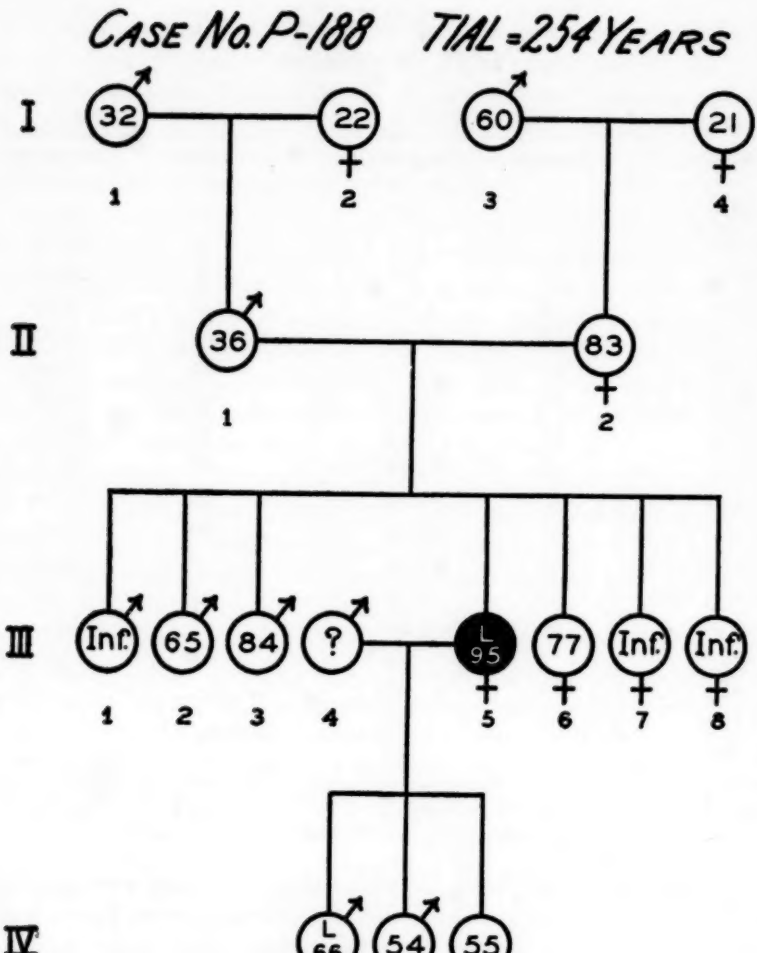

FIG. 9. PEDIGREE OF INDIVIDUAL HAVING LOWEST OBSERVED TIAL

In this and the following pedigree the propositus is indicated by a solid sex sign. Figures within the circles of the sex signs indicate *ages at death in years,* except where there is an L above the age figure, which means that the person was living at the time of record, and at the indicated age in years. In this case the propositus was living at age 95, the only survivor of a sibship of seven, three of whom died in infancy (III, 1, 7 and 8). Only one brother (III, 3) achieved anything especially remark-

the FHR Series. In both series of observations the highest observed TIAL is well over twice as large as the lowest observed. In other words the summed duration of life of one person's parents and grandparents taken together may well be more than double that of another person, whether the person is a nonagenarian or only just mediocre in respect of his own attained longevity.

The distributions of Table 9 have been fitted, by the method of moments, with Pearsonian frequency curves. The constants are given in Table 10.

TABLE 10

Biometric Constants of the TIAL Distributions in Table 9

CONSTANT	LONG. SERIES	FHR SERIES
Mean	446.14 ± 1.66 yrs.	385.45 ± 2.65 yrs.
Median (from observation)	452.10 ± 2.08 yrs.	382.40 ± 3.32 yrs.
Mode	454.18 ± 2.03 yrs.	Same as mean
Standard deviation	46.88 ± 1.17 yrs.	47.02 ± 1.88 yrs.
Coefficient of variation	10.52 ± .26%	12.20 ± .49%
μ_2	5.4948	5.5270
μ_3	— 5.2736	— .4515
μ_4	113.6927	75.0338
β_1	.1755	.0012
β_2	3.7814	2.4563
Skewness	—.173 ± .043	...
Curve Type	IV	II

able in longevity, although a sister (III, 6) died only three years short of 80. Two of the three children of the propositus died in their fifties. Of the six immediate ancestors of the propositus only two (I, 3, and II, 2) lived past fifty, and only one, her mother (II, 2) could be regarded as really longevous. The causes of death of the ancestors were as follows: II, 1, smallpox; II, 2, "senility," that is to say, cause of death unknown; I, 1, "fever"; I, 2, in childbirth; I, 3, "rheumatism"; I, 4, pulmonary tuberculosis following childbirth.

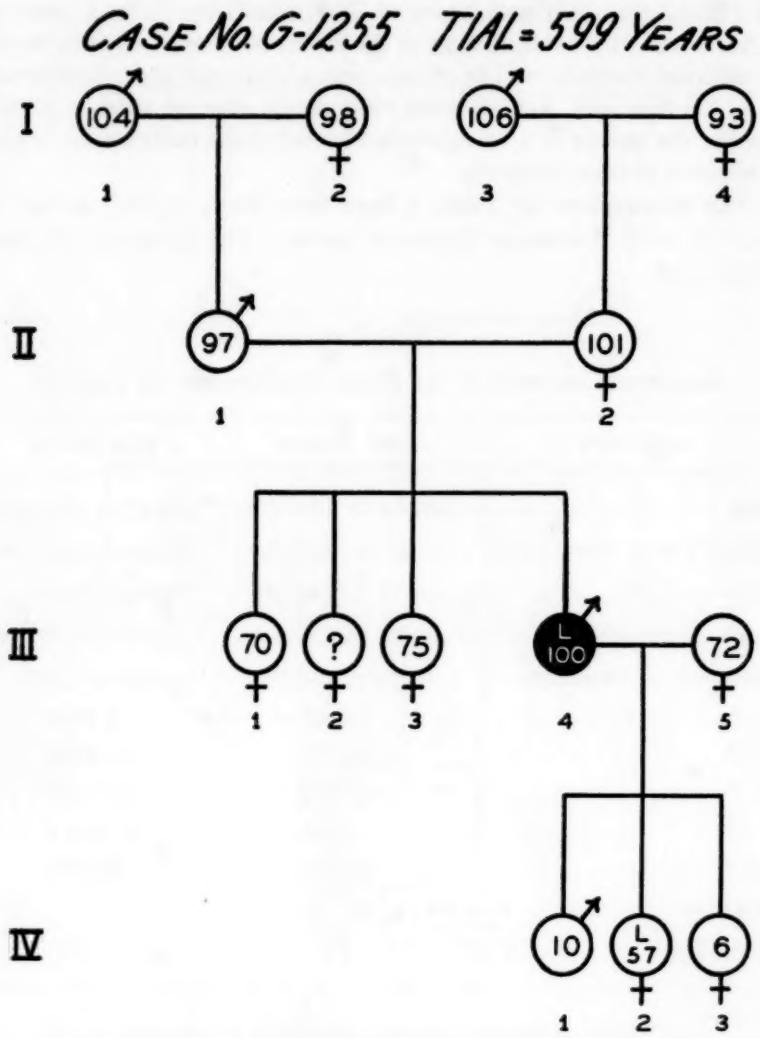

FIG. 10. PEDIGREE OF INDIVIDUAL HAVING HIGHEST OBSERVED TIAL

In this case the propositus (III, 4), living at the age of 100, was a Scottish seafaring man, who married and "settled down" at the age of 39. His immediate ancestry is very remarkable in point of longevity. His father (II, 1) and his paternal grandmother (I, 2) died as the result of accidents. The causes of death of his other immediate ancestors are unknown. The two children (IV, 1 and IV, 3) died of drowning.

The histograms and fitted curves of the two distributions are shown in Figure 11. In this diagram relative (percentage) frequencies are plotted. The equations for the fitted curves are:

Long. Series $$y = 6.8802 \left(1 + \frac{x^2}{86.4231}\right)^{-11.4393} e^{-10.3301 \tan^{-1} \frac{x}{9.2964}}$$

Origin at 538.1285 years

FHR Series $$y = 22.163 \left(1 - \frac{x^2}{49.9374}\right)^{3.0176}$$

Origin at mean

In these equations y denotes absolute frequency and x denotes years.

Figure 11 makes plain the great difference in the TIAL distribution in the two series. By no chance could they be looked upon as random samples from a single population in respect of TIAL.

The difference between the two series is shown graphically in another way in Figure 12. In that diagram the two fitted curves of Figure 11 are plotted in the cumulated, or so-called integral form, showing the TIAL values possessed by successive rising percentages of the individuals in each series. Thus the 10 per cent of the FHR Series having the lowest TIAL values show TIALs below 318 years, roughly, as contrasted with 383 years, roughly, for the 10 per cent of the Long. Series with the poorest ancestral longevity. The heavy horizontal lines between the curves at each 10 per cent level show, when read against the abscissal axis, the number of years by which the TIAL of the indicated percentage of the Long. Series exceeds that for the same percentage of the FHR Series.

From the data so far presented it appears that the mean TIAL for the group of nonagenarians and centenarians is 60.69 ± 3.13 years, or 15.75 per cent *greater* than for the FHR Series not directly selected relative to age. The difference is 19.3 times its probable error, and therefore far greater than could reasonably be expected to arise by a chance fluctuation of sampling. It amounts to something more than one average lifetime. At first sight it may perhaps seem puzzling that the difference between the TIALS of the two series is not even greater, considering the stringent selection implied in the low frequency of nonagenarians in the living population. It must, however, be remembered that the propositus in the FHR Series is living, in each case except 7 out

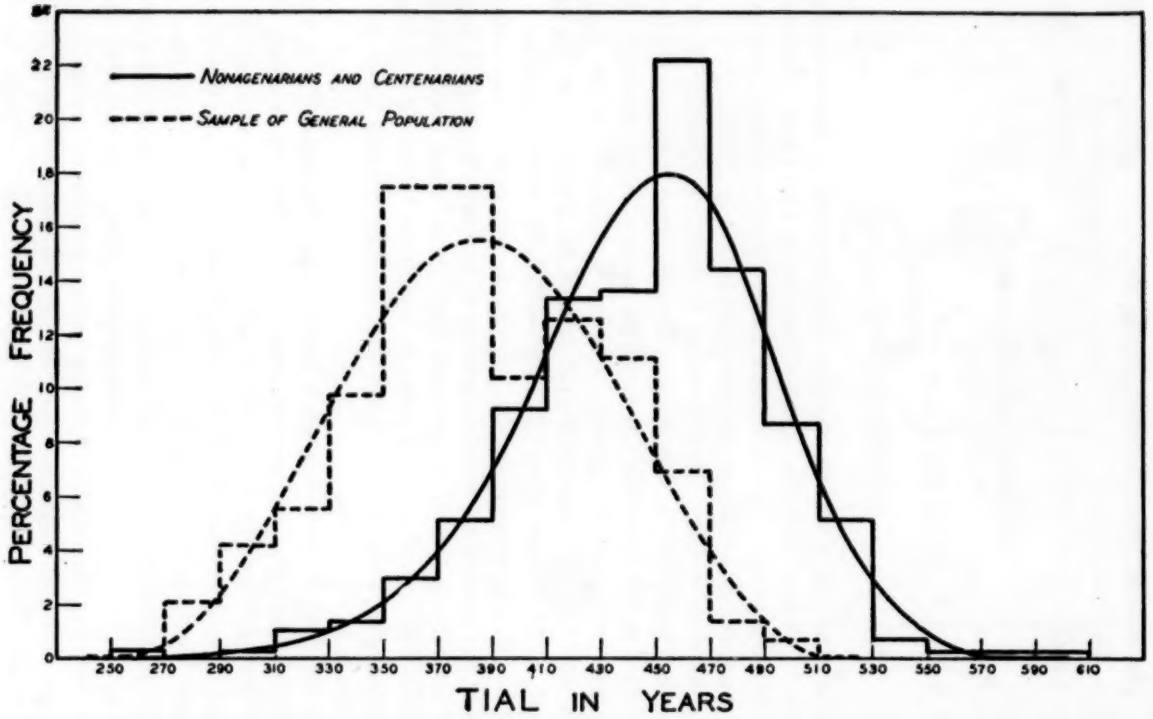

FIG. 11. HISTOGRAMS AND FITTED CURVES SHOWING THE DISTRIBUTION OF VARIATION IN TIAL FOR (A) NONAGENARIANS AND CENTENARIANS (LONG. SERIES), AND (B) COMPARISON GROUP (FHR SERIES)

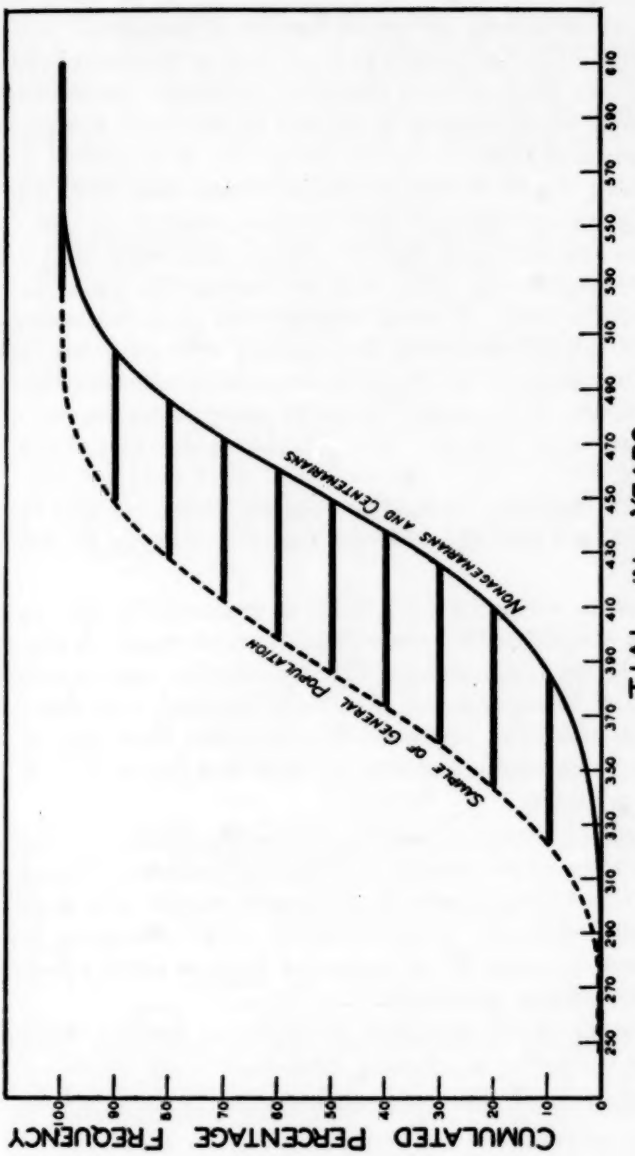

FIG. 12. CUMULATED (INTEGRAL) CURVE OF VARIATION IN TIAL IN THE LONG. SERIES AND FHR SERIES. FOR FURTHER EXPLANATION SEE TEXT

of the 143. In these 7 cases there were no living members of the offspring sibship from which the propositus had to be taken, so the sibling dead at the highest age played the rôle of propositus. Since the propositi in the FHR Series were persons living at the time of record it follows that they went on living thereafter, and some fraction of them may reasonably be expected to be by way of becoming nonagenarians with the passage of time.

In the Long. Series one-half of the individuals have TIALs of less than 452.1 years, and one-half have TIALs greater than that figure. The corresponding figure for the FHR Series, 382.4 years, is 69.7 ± 3.9 years lower—considerably more than an average life time, and 17.9 times its probable error. It must, however, always be remembered that some part of this difference may be accounted for by the fact that the manner of formation of the FHR Series automatically tends to lower the TIAL values. This effect will chiefly apply to the parents of the propositi, and only slightly (and probably insignificantly) to the grandparents.

The most frequently occurring (modal) TIAL is 68.73 ± 3.33 years, or 17.8 per cent higher in the Long. Series than in the FHR Series.

The absolute variability of TIAL, as measured by the standard deviation, is insignificantly greater (0.14 ± 2.21 years) in the FHR Series than in the Long. Series. The difference in relative variability as measured by the coefficient of variation is, however, somewhat larger, amounting to $1.68 \pm .55$ per cent. This is on the borderline of probable statistical significance, and is in the same direction as the difference in absolute variability.

The degree of relative variability exhibited in TIAL (coefficient of variation of 10 to 12 per cent) is moderate in the scale of human characters generally.[18] It compares in this respect roughly with such characters as body weight, skin pigmentation of whites, internipple breadth, height of mandible, relative cell volume of blood in active tuberculosis, lower nasal breadth in whites, etc.

Careful study of the data gives no indication that the variation in TIAL, in either series, is anything other than really continuous. In samples of the present size the irregularities in the frequency distribu-

[18] See Table 57 (pp. 347-349) of Pearl, R., Introduction to Medical Biometry and Statistics. Second Edit. *Philadelphia* (W. B. Saunders Co.), 1930.

tion cannot, in our opinion, be regarded as indicative of any real discontinuity in variation. By appropriate mathematical juggling we could graduate either of the TIAL distributions as the sum of two skew frequency curves just as could probably be done with practically any other frequency distribution whatsoever, but so far as any existing evidence goes the resultant would be only an expression of smart mathematical practice, and not in the least a description of a real natural phenomenon.

B. Contribution to TIAL from Paternal and Maternal Sides of the Pedigree

Having examined the variation in TIAL as a whole we may now proceed to its analysis. TIAL may be divided in various ways. Of these the following will be considered first:

$$\text{TIAL} = (a + b + e) + (c + d + f).$$

$(a + b + e)$ is the contribution to TIAL from the paternal side and $(c + d + f)$ the contribution from the maternal side.

Table 11 gives the biometric constants for these two moieties.

TABLE 11

Biometric Constants for Paternal and Maternal Moieties of TIAL

CONSTANT	LONG. SERIES		FHR SERIES	
	Paternal $(a+b+e)$ (*years*)	Maternal $(c+d+f)$ (*years*)	Paternal $(a+b+e)$ (*years*)	Maternal $(c+d+f)$ (*years*)
Mean	223.48±1.14	223.26±1.06	196.71±1.68	188.04±1.84
Standard deviation ...	32.34± .81	30.11± .75	29.82±1.19	32.54±1.30
Coefficient of variation ...	13.85± .35	12.72± .32	15.16± .62	17.31± .71

It will be noticed at once that the sums of the mean paternal and maternal components of TIAL in Table 11 do not exactly equal the means and medians of the total TIALs in Table 10. The discrepancies arise because of the effects of grouping the frequency in 20-year classes in the computations. In making Table 11 the actual recorded ages of

$(a + b + e)$ were separately summed for each individual propositus. These sums were then put into a frequency distribution and the individuals in a class were assumed, as usual, to center at the mid-point of that class. The maternal component was dealt with in the same way. Discrepancies of the sort observed may be expected to arise under this procedure, even though the computations are accurate at all stages. In order to make clear exactly what these computational discrepancies amount to in Table 11, and subsequent tables, we insert here Table 12. This table shows (a) the mean values got from a combined frequency distribution of components of TIAL, (b) the sums of the separately computed means of the several components, and (c) the differences between the two and their probable errors. The differences are taken as + when the sum of the mean of the components (b) is the larger value.

It is clear from Table 12 that the discrepancies between the summed means and the means computed from combined frequencies are in no case statistically significant in either series.

Having now cleared up this technical point we may return to the consideration of the relative contributions to TIAL from the paternal and maternal sides of the pedigrees as set forth in Table 11.

Of the relationships shown by the data of Table 11 perhaps the most striking is that whereas in the Long. Series the contributions to TIAL from the paternal side of the pedigree and from the maternal side (whether measured by means or medians) are identical within less than 4 months in a total of over 200 years, the same is not true for the FHR Series. In the FHR Series the contribution to TIAL from the paternal side is distinctly larger than that from the maternal. The difference in the mean contribution is $196.71 - 188.04 = 8.67 \pm 2.49$ years. The odds against this difference being a chance fluctuation of sampling are nearly 54 to 1. It may therefore be concluded that in this sample the immediate ancestors on the maternal side are probably definitely inferior in longevity to those on the paternal side, in the case of the FHR Series, the group of ordinary longevity.

The differences between the Long. Series and the FHR Series in respect of the mean contribution to TIAL from the paternal and maternal sides are:

Paternal; $223.48 - 196.71 = 26.77 \pm 2.03$ years
Maternal; $223.26 - 188.04 = 35.22 \pm 2.12$ years

TABLE 12

Different Methods of Computing Mean Values of TIAL and Its Components

COMPONENTS OF TIAL	LONG. SERIES			FHR SERIES		
	Mean computed from frequency distribution of summed observations (a)	Sum of means of components (b)	Difference (c)	Mean computed from frequency distribution of summed observations (a)	Sum of means of components (b)	Difference (c)
$e+f=$ Parental component	144.63	145.75	+1.12±1.27	114.34	115.66	+1.32±2.11
$a+b+c+d=$ Grandparental component	301.34	306.05	+4.71±1.71	270.14	274.27	+4.13±2.79
$(a+b+c+d)+(e+f)=$ TIAL ...	446.14	445.97	− .17±2.31	385.45	384.48	− .97±3.73
$a+b+e=$ Paternal component	223.48	226.22	+2.74±1.51	196.71	199.19	+2.48±2.30
$c+d+f=$ Maternal component	223.26	225.58	+2.32±1.46	188.04	190.74	+2.70±2.50
$(a+b+e)+(c+d+f)=$ TIAL ...	446.14	446.74	+ .60±2.28	385.45	384.75	− .70±3.64
$a+c+e=$ Male component	222.71	224.91	+2.20±1.47	195.17	197.24	+2.07±2.29
$b+d+f=$ Female component	223.97	226.89	+2.92±1.48	190.14	192.69	+2.55±2.38
$(a+c+e)+(b+d+f)=$ TIAL ...	446.14	446.68	+ .54±2.26	385.45	385.31	− .14±3.55
$a+b+c+d+e+f=$ TIAL	446.14	451.80	+5.66±2.18	385.45	389.93	+4.48±3.52

These are obviously significant differences. The immediate ancestors on the paternal side of this group of nonagenarians and centenarians show about 14 per cent more accumulated longevity than do the corresponding ancestors of the FHR group. On the maternal side the superiority amounts to about 19 per cent.

There appears to be no definitely significant difference in variability of the contributions to TIAL from either the paternal or the maternal side. This is true of both the Long. and the FHR groups.

C. Contributions to TIAL from Male and Female Ancestors

How does the contribution to TIAL from the three male immediate ancestors compare with that from the three female? Here we have

$$\text{TIAL} = (a + c + e) + (b + d + f).$$

The data are given in Table 13.

The three immediate male ancestors in the Long. Series have a mean summed longevity of 27.54 ± 1.99 years, or 14.1 per cent greater than that shown by the corresponding male ancestors in the FHR Series. The mean excess in longevity of the three immediate female ancestors in the Long. Series over the corresponding ancestors in the FHR Series is 33.83 ± 1.99 years, or 17.8 per cent.

In the Long. Series the female immediate ancestors show only a small superiority, insignificant statistically, over the males in summed longevity. One would expect mothers and grandmothers to be definitely older at death, on the average, than fathers and grandfathers, because of the general superiority of females over males in life duration, which is one of the earliest and best established generalizations of biostatistics. The present results suggest, though it would be hazardous to conclude, that in the immediate ancestry of extremely longevous persons the usual rule as to the sex difference in longevity does not hold with its usual force. More data than are at present available would, however, be required to establish such an exception.

In the FHR Series it appears that the immediate female ancestors as a group are inferior in summed longevity to the immediate male ancestors. The difference in the means amounts to 5.03 ± 2.36 years, which cannot be regarded as statistically significant.

In neither series of data is there any significant difference in variability, either absolute or relative, in the summed longevity of male as compared with female immediate ancestors.

TABLE 13

Biometric Constants for Male and Female Moieties of TIAL

CONSTANTS	LONG. SERIES		FHR SERIES	
	Male ancestors $(a+c+e)$ (*years*)	Female ancestors $(b+d+f)$ (*years*)	Male ancestors $(a+c+e)$ (*years*)	Female ancestors $(b+d+f)$ (*years*)
Mean	222.71 ± 1.09	223.97 ± 1.08	195.17 ± 1.67	190.14 ± 1.67
Standard deviation	30.89 ± .77	30.71 ± .77	29.51 ± 1.18	29.68 ± 1.18
Coefficient of variation	13.02 ± .33%	13.71 ± .33%	15.12 ± .62%	15.61 ± .64%

D. Generational Contributions to TIAL

A third way in which TIAL may be broken up is

$$\text{TIAL} = (a + b + c + d) + (e + f),$$

which will give separately the total grandparental and parental contributions.

Table 14 presents the biometric constants for this division of the material.

The four grandparents in the Long. Series exhibit a mean summed longevity 31.20 ± 2.45 years, or 11.5 per cent, in excess of that of the four grandparents in the FHR Series. Here again, just as in each case so far examined, it is seen that however TIAL is broken up into its components the superiority of the group of nonagenarians and centenarians over the FHR group (individuals not directly selected for longevity) in respect of ancestral longevity is marked and significant. As has already been pointed out, this grandparental difference between the two series cannot be accounted for in any significant degree by the manner of selection of the two series.

The two parents in the Long. Series have a mean summed longevity of 30.29 ± 1.86 years, or 26.5 per cent greater than that of the parents in the FHR Series.

Here we encounter for the first time a relationship which will reappear in various forms as we proceed. While there are but two parents as against four grandparents contributing to TIAL the excess of the mean summed longevity in the Long. Series as compared with the FHR Series is *absolutely* almost identically the same for the parental contribution as it is for the grandparental, and therefore relatively about twice as large. In short, it appears that high parental longevity is more important to the individual than high grandparental longevity in his own quest for the achievement of great age.

If each one of the six immediate ancestors made exactly equal contributions to TIAL the mean total contribution of the grandparents in the Long. Series would be two-thirds of the total, or 297.43 years. Actually the mean grandparental contribution was 301.34 years, or 3.91 years in excess of expectation on an equal contribution basis. On the same assumption the expected mean parental contribution in the Long. Series would be 148.71 years. The actual was 144.63, or 4.08 years less. In both cases these differences are small, indeed so small that,

TABLE 14

Biometric Constants for Grandparental and Parental Contributions to TIAL

CONSTANTS	LONG. SERIES		FHR SERIES	
	Grandparents $(a+b+c+d)$ (*years*)	Parents $(e+f)$ (*years*)	Grandparents $(a+b+c+d)$ (*years*)	Parents $(e+f)$ (*years*)
Mean	301.34 ± 1.31	144.63 ± .92	270.14 ± 2.07	114.34 ± 1.62
Standard deviation	37.04 ± .92	26.02 ± .65	36.77 ± 1.47	28.69 ± 1.14
Coefficient of variation	12.29 ± .31%	17.99 ± .46%	14.70 ± .60%	25.09 ± 1.06%

having regard to the probable errors involved, the conclusion would seem to be that *in the Long. Series* the grandparents and the parents, as groups, contributed so nearly their respective two-thirds and one-third to TIAL as to make no matter practically.

The case is different in the FHR Series. There the expected grandparental contribution to TIAL, on the assumption of equal individual ancestral longevity, is 256.97 years. The actual is 270.14 years, an excess over expectation of 13.17 years. The expected parental contribution of one-third of TIAL is 128.48 years. The actual parental contribution is 114.34 years, an amount 14.14 years in defect of expectation. In other words, in the FHR Series the parents were relatively short lived. A part of this is probably a purely statistical consequence of the manner in which the group was formed, but exactly how much cannot be determined from the available data.

E. Individual Contributions to TIAL

Table 15 gives the biometric constants for the six individual ancestors whose combined contributions make up TIAL, that is where

$$\text{TIAL} = a + b + c + d + e + f.$$

The first thing that strikes one in examining Table 15 is the great regularity of the constants for the ages at death of the six immediate ancestors in both series. The mean ages of the four grandparents, in each series, agree with each other to a degree well within the fluctuations of random sampling. The same is true of their standard deviations and coefficients of variation. The same thing is also true of the parents in the Long. Series, but not in the FHR Series.

The individual ancestors in the Long. Series have higher mean ages at death, and lower coefficients of variation, in each case, than do the corresponding ancestors in the FHR Series. The standard deviations in the two series are plainly not significantly different from each other. The absolute and relative differences in the mean, together with the probable errors, and the absolute differences and probable errors of the coefficients of variation are shown in Table 16.

Each of the four grandparents in the Long. Series had an average duration of life from 10 to 14 per cent (11.6 average) longer than the corresponding ancestor in the FHR Series. These differences are certainly significant, being about 7 times their probable errors. The dif-

TABLE 15

Biometric Constants for Age at Death of the Six Immediate Ancestors of the Propositus in the Long. Series and the FHR Series

ANCESTRY	MEAN		STANDARD DEVIATION		COEFFICIENT OF VARIATION	
	Long. Series (*years*)	FHR Series (*years*)	Long. Series (*years*)	FHR Series (*years*)	Long. Series (*per cent*)	FHR Series (*per cent*)
a. Father's father ...	76.75± .56	68.99± .92	15.73± .39	16.35± .65	20.49± .53	23.70±1.00
b. Father's mother ..	77.05± .56	70.10± .93	15.87± .40	16.55± .66	20.60± .54	23.61± .99
c. Mother's father ..	75.74± .55	68.15± .93	15.64± .39	16.40± .65	20.65± .54	24.07±1.01
d. Mother's mother ..	76.51± .54	67.03± .96	15.43± .39	17.08± .68	20.16± .52	25.48±1.07
e. Father	72.42± .60	60.10± .87	16.86± .42	15.37± .61	23.28± .61	25.57±1.08
f. Mother	73.33± .64	55.56±1.04	18.03± .45	18.39± .73	24.59± .65	33.10±1.46

TABLE 16

ANCESTRY	EXCESS OF LONG. MEAN OVER FHR MEAN (*years*)	PERCENTAGE EXCESS	DIFFERENCE IN RELATIVE VARIABILITY (LONG. C. OF V) — (FHR C. OF V.)
a. Father's father	7.76 ± 1.08	11.2	—3.21 ± 1.13
b. Father's mother	6.95 ± 1.09	9.9	—3.01 ± 1.13
c. Mother's father	7.59 ± 1.07	11.1	—3.42 ± 1.15
d. Mother's mother ...	9.48 ± 1.10	14.1	—5.32 ± 1.15
e. Father	12.32 ± 1.06	20.5	—2.29 ± 1.24
f. Mother	17.77 ± 1.22	32.0	—8.51 ± 1.60

ferences in grandparental coefficients of variation, the FHR Series being the more variable in each case, can only be regarded as probably significant statistically. Except in one case (mother's mother) they are just under three times the probable error.

The excess longevity of each of the parents in the Long. Series as compared with the FHR Series is much larger than the grandparental excess, in fact nearly double.

The meaning of the data set forth in Tables 15 and 16 will perhaps be made clearer by an examination of Figures 13 to 15, which put the results graphically in the form of pedigrees. All three of these diagrams are drawn on the same plan. Figure 13 deals with mean ages. In the left-hand panel the pedigree gives, by the figures within the sex signs, the mean age at death of each ancestor in the Long. Series, and within the rectangular box at the bottom (a) the mean living ages of the propositi, and (b) the expected mean total longevities when they are all dead. The pedigree in the right hand panel does the same thing for the FHR Series. In the middle panel the pedigree shows in the same way the differences, both absolute (upper figures, years) and relative (lower figures, percentages) between the Long. and the FHR Series. The percentages are taken as of the difference to the smaller component entering into it.[18a]

[18a] By an inadvertent error in drafting, first discovered in the proof, the percentage 37.6 at the bottom of the middle panel of Fig. 14 is incorrect. It should be 36.0.

FIG. 13. INFLUENCE OF IMMEDIATE ANCESTORS UPON MEAN (AVERAGE) LONGEVITY
FOR FURTHER EXPLANATION SEE TEXT

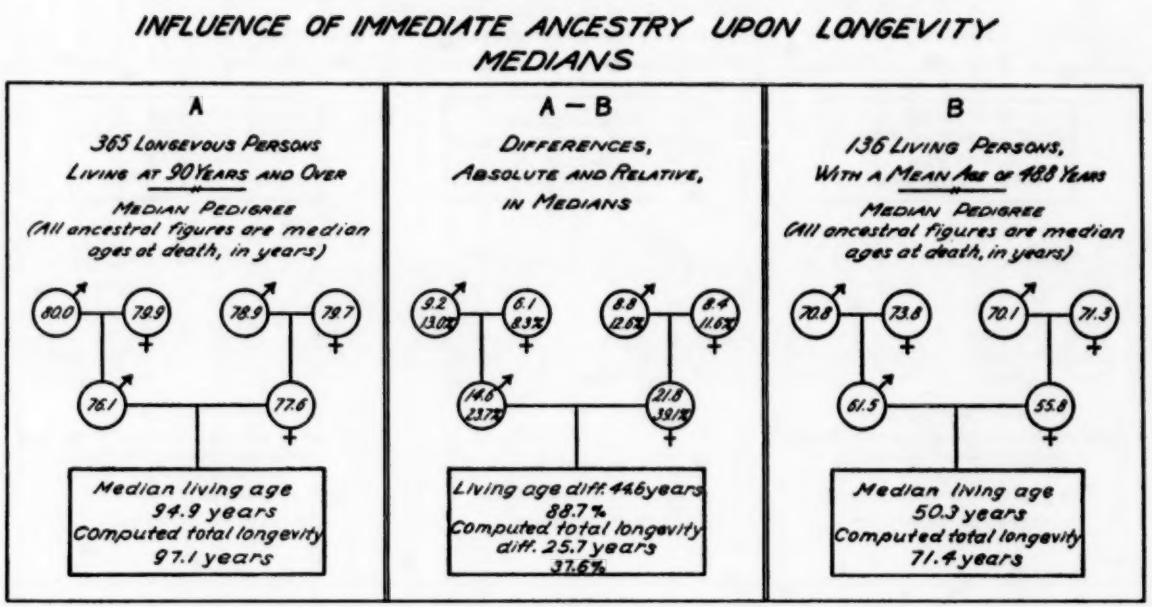

FIG. 14. INFLUENCE OF IMMEDIATE ANCESTRY UPON MEDIAN LONGEVITY

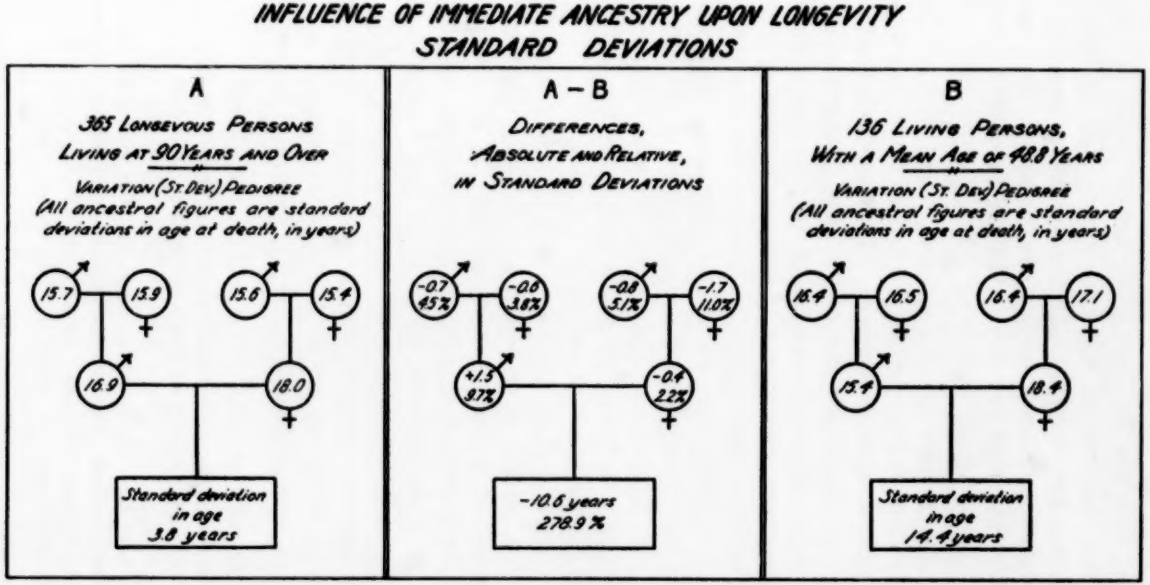

Fig. 15. Variation in Ancestral Longevity

Comparing pedigree A of Figure 13 with pedigree B leads to the following broad general result: We select two groups of living persons (the propositi) such that the mean living age of one group (A) is 95.5 per cent greater than that of the other group. On its face this is a stringent selection for diversity. But the *parents* of these two diverse groups differ in completed longevity not by 95 per cent, but by an average of only about 26 per cent. And their *grandparents* differ in completed longevity by an average of only 11.6 per cent. To put it another way the parents of two groups widely divergent in mean realized longevity up to the time of record differ rather less than a third as much in their completed longevity. It must, however, be clearly recognized that a considerable part of the great divergence between the propositi of the two series in mean living age arises from the fact that it is *living* age under discussion. If we put in place of mean living age the expected mean *final* longevities (realized plus expected from the life table) as computed earlier we find the computed total longevities of the propositi (and the resultant differences) given at the bottom of the panels of Figures 13 and 14. They show that even upon the basis of completed longevity the propositi of the two series are more different from each other than are their respective parents.

The two groups of grandparents are less divergent from each other in longevity than are the parents—are in fact plainly quite close to the average longevity of grandparents in general. If it were possible to get the data for a great-grandparental generation we should probably find, in samples of the order of size of these here dealt with, that each of the two sets of great-grandparents were practically random samples of great-grandparents in general, and indistinguishable from each other.

This result is, of course, merely one particular example of a general rule, which has been strikingly stated by Pearson[19] in the following passage:

> Twenty generations ago each one of us, but for cousin or other blood marriages, would have over a million ancestors of the same generation, and that is only 600 to 700 years ago. You will see that the ancestors of any one of you must 1000 years ago have been nearly synonymous with the total population of Great Britain and therefore their average was mediocre or your mid-ancestor of that generation was identical with the type of the inhabitants of this country or rather of a still wider range of mankind. I have endeavored to follow back the

[19] Pearson, K. On a new theory of progressive evolution. *Ann. of Eug.*, Vol. 4, pp. 1-40, 1930.

ancestry of Charles Darwin and in those lines in which I can follow it, I find that a thousand years ago his known ancestors were scattered over the whole face of Europe, and stretched over Russia to Persia and through Constantinople to Asia Minor. No escape from this burden of ancestry is possible either in man or beast except by isolation and inbreeding.

Let us examine a little more closely the 10th generation of ancestry; we each had 1024 ancestors, between 300 and 400 years ago. Their average must be very nearly mediocrity, or type, for 1000 is a fair sized sample anthropometrically, as you would appreciate if you attempted to measure 30 or 40 characters in 1000 skulls! Indeed I may bring it still further home, for it would be difficult to find a man with 16 exceptional great-great-grandparents, and the average even of 16 individuals, if they are selected at random, will not be very different from mediocrity for any single character.

Figure 14, constructed on the same plan as Figure 13, but for median ages, gives the same general result as we have seen from the means.

Figure 15, in which the pedigrees are for absolute variability as given by the standard deviation, shows that the great difference in this respect between the propositi in the Long. and the FHR Series in respect of living age diminishes to very small and statistically insignificant amounts in the parental and grandparental generations. Such ancestral differences in variability as there are, however, are all of the same sign except in the case of the fathers. The ancestry of the individuals in the FHR Series is, with the exception noted, consistently slightly more variable in life duration than that of the individuals in the Long. Series.

Let us now consider some comparative results. The question of the mean age at death, for example, of particular ancestors (say fathers or grandfathers) is an extremely interesting one, and one about which comparatively little is known. Similarly one would like to know something of the variability in longevity of ancestors. It will therefore be useful to bring together in a comprehensive table the available data upon these points. This is done in Table 17, which contains means, standard deviations, and coefficients of variation for age at death of parents and grandparents. Data regarding more remote ancestors have apparently not been published.

In computing the average figures at the end of each section of Table 17, to serve as a first approximation to norms until more and better data can be added to what is here tabulated, the following procedure was followed. For the unweighted averages (1) the *means* for cases where *N*, mean and S. D. were given were added and divided by

the number of such cases; (2) the *standard deviations* for the same cases were added and divided by the number of such cases; (3) the mean standard deviation as described under (2) was multiplied by 100 and divided by the average mean computed as described under (1) to get the average *coefficient of variation*; (4) to get the probable errors of the averaged constants the average S. D. (2) and the average C. of V. (3) were used, with the average N of the cases used. The procedure in the case of the weighted average constants was the same except that each constant was weighted in averaging with its N as tabled, but in computing the probable errors the same average N as in (4) was used. This is a conservative procedure. As a matter of fact there is but little difference between the weighted and unweighted average constants in any case. For grandparents no average constants are given because the material is too meager to make such a procedure of any value.

From an examination of Table 17, which summarizes essentially all the knowledge of the longevity of ancestors in man that has been gained by the application of biometric methods to pedigree data, the following points worthy of note emerge:

1. The mean age at death of fathers ranges from 54.33 years for fathers of living tuberculous persons to 72.42 years for fathers of nonagenarians and centenarians, a range of 18.09 years. This is more than twice as great a range as is found for grandfathers, where the extremes are 68.15 years for maternal grandfathers in the FHR Series and 76.75 years for paternal grandfathers of nonagenarians and centenarians (Long. Series), a range of 8.60 years. But the available data for grandfathers are very meager. Presumably as further material from other differentiated samples becomes available the range of mean ages at death of grandfathers will widen somewhat. But it also must be remembered that there is some reason to believe that grandfathers in general will always prove to be a more homogeneous class of human beings in respect of age at death than parents; primarily because, as has already been suggested, they tend to be more nearly a random sample of the whole population in respect of genetic constitution. Even with the meager data available the coefficients of variation of Table 17 suggest that fathers tend slightly to be more variable individually in age at death than grandfathers, but the material available for comparison is so small that this can be no more than a tentative suggestion.

TABLE 17

Biometric Constants for Age at Death of Various Ancestors

A. Fathers

ITEM NO.	AUTHORITY	NATURE AND SOURCE OF MATERIAL	KIND OF PEOPLE	N	MEAN (*years*)	STANDARD DEVIATION (*years*)	COEFFICIENT OF VARIATION (*per cent*)
1	Pearl, this study, Table 15	Fathers of nonagenarians and centenarians	Chiefly American	365	72.42± .60	16.86± .42	23.28± .61
2	Stoessiger,[26] Table I	Pearson Family Data. Accidents and infections omitted. Fathers of sons.....	British	892[21]	71.33± .29	12.87± .21	18.04[22]
3	Pearl,[24] Table 2	Fathers of persons living at age of 90 and over. Nonaccidental causes of death.....	American	37	71.15±2.01	.....	.....
4	Stoessiger,[26] Table I	Pearson Family Data. Inclusive. Fathers of sons.....	British	1032[21]	70.47± .28	13.40± .20	19.02[22]
5	Stoessiger,[26] Table I	Pearson Family Data. Inclusive. Fathers of daughters...	British	911[21]	69.90± .31	13.98± .22	19.99[22]
6	Pearl,[23] Table VII	Family History Records. Fathers of selected noncancerous persons	Urban American	120	69.83± .97	15.76± .69	22.57[22]
7	Beeton and Pearson,[20] Table A ...	Quaker genealogies. Fathers of adult daughters.....	British	1156[21]	69.55± .27	13.66± .19	19.65[22]
8	Beeton and Pearson,[20] Table A ...	Quaker genealogies. Fathers of adult sons	British	1000[21]	68.37± .31	14.70± .22	21.50[22]
9	Wilson and Doering,[27] p. 428	Peirce Genealogy. Fathers with consort's age at death known..	American	135	68.0 ±1.3	.....	.....

TABLE 17—*Continued*

A. Fathers—Continued

ITEM NO.	AUTHORITY	NATURE AND SOURCE OF MATERIAL	KIND OF PEOPLE	N	MEAN (*years*)	STANDARD DEVIATION (*years*)	COEFFICIENT OF VARIATION (*per cent*)
10	Beeton and Pearson,[27] p. 298	Landed Gentry. Fathers of sons dying at 50 years and over	British	?[21]	66.88	14.33	21.42[22]
11	Beeton and Pearson,[27] p. 298	Peerage. Fathers of sons dying at 52.5 years and over.....	British	?[21]	66.68	14.67	22.01[22]
12	Pearl,[23] Table VII	Family History Records. Fathers of cancerous persons	Urban American	120	66.33± .90	14.66± .64	22.10[22]
13	Wilson and Doering,[27] p. 428	Peirce Genealogy. All fathers	American	187	66.2 ±1.1	.....	.....
14	Beeton and Pearson,[27] p. 297	Landed Gentry. Fathers of sons dying at 20 yrs. and over	British	1000[21]	65.96	14.43	21.88[22]
15	Beeton and Pearson,[27] p. 297	Peerage. Fathers of sons dying at 25 years and over.....	British	1000[21]	65.84	14.64	22.23[22]
16	Beeton, Yule and Pearson,[28] Table IV	Burke's Landed Gentry. Fathers weighted with their fertility	British	5336[30]	65.51	.....	.....
17	Bell,[31] p. 50	Hyde Genealogy	American	795	65.0	.....	.....
18	Beeton and Pearson,[20] Table A	Quaker genealogies. Fathers of minor sons	British	943[21]	63.85± .33	15.18± .24	23.78[22]
19	Beeton, Yule and Pearson,[28] Table I	Burke's Landed Gentry. Age at death of father, correlated with size of family.....	British	1000	63.58	15.19	23.88[22]
20	Beeton and Pearson,[20] Table A ...	Quaker genealogies. Fathers of minor daughters	British	853[21]	63.57± .37	15.84± .26	24.92[22]

	son,[20] Table A ...	minor daughters	British	853 [21]	63.57± .37	15.84± .26	24.92 [22]

TABLE 17—*Continued*

A. Fathers—Continued

21	Pearl,[23] Table III	Family History Records. Fathers of selected dead nontuberculous persons	Urban American	504	63.51± .48	16.07± .34	25.30 [22]
22	Beeton, Yule, and Pearson,[28] Table IV	Whitney Genealogy chiefly. Fathers weighted with their fertility	American	5928 [29]	63.08	.....	.....
23	Pearl,[23] Table III	Family History Records. Fathers of dead tuberculous persons	Urban American	504	61.35± .49	16.32± .35	26.60 [22]
24	Gould,[25] Fig. 1 ...	Genealogical material. "Fathers not grandfathers"	American	? (ca. 50)	60.68±1.56	.....	.....
25	Pearl, this paper, Table 15	Fathers in FHR Series.....	Urban American	143	60.10± .87	15.37± .61	25.57±1.08
26	Beeton, Yule, and Pearson,[28] Table I	Whitney Genealogy chiefly. Age at death of fathers correlated with size of family.....	American	1084	58.09	16.07	27.66 [22]
27	Pearl,[23] Table III	Family History Records. Fathers of selected living nontuberculous persons	Urban American	180	57.17± .81	16.05± .57	28.07 [22]
28	Pearl,[23] Table III	Family History Records. Fathers of living tuberculous persons	Urban American	180	54.33± .75	14.95± .53	27.52 [22]
29	Unweighted averages for fathers, including all (and only) cases for which *N*, mean, and S.D. are given.....			683.5	65.03± .39	15.05± .27	23.15± .44
30	Weighted averages for fathers, including all (and only) cases for which *N*, mean, and S.D. are given.....			12,987 [32]	65.91± .38	14.78± .27	22.43± .43

TABLE 17—*Continued*

B. Mothers

ITEM NO.	AUTHORITY	NATURE AND SOURCE OF MATERIAL	KIND OF PEOPLE	N	MEAN (*years*)	STANDARD DEVIATION (*years*)	COEFFICIENT OF VARIATION (*per cent*)
31	Pearl, this study, Table 15	Mothers of nonagenarians and centenarians	Chiefly American	365	73.33± .64	18.03± .45	24.59± .65
32	Pearl,[24] Table 2	Mothers of persons living at ages of 90 and over. Non-accidental causes of death	American	44	72.73±1.73	.....	.....
33	Stoessiger,[26] Table I	Pearson Family Data. Inclusive. Mothers of daughters	British	796[21]	71.72± .34	14.26± .24	19.88[22]
34	Stoessiger,[26] Table I	Pearson Family Data. Accidents and infections omitted. Mothers of sons	British	819[21]	70.86± .36	15.19± .25	21.44[22]
35	Stoessiger,[26] Table I	Pearson Family Data. Inclusive. Mothers of sons	British	916[21]	70.57± .34	15.22± .24	21.56[22]
36	Beeton and Pearson,[20] Table A	Quaker genealogies. Mothers of adult daughters	British	1064[21]	68.70± .34	16.42± .24	23.90[22]
37	Beeton and Pearson,[20] Table A	Quaker genealogies. Mothers of adult sons	British	1220[21]	67.95± .33	16.90± .23	24.88[22]
38	Pearl,[23] Table VII	Family History Records. Mothers of cancerous persons	Urban American	107	67.71±1.07	16.36± .75	24.16[22]
39	Pearl,[23] Table VII	Family History Records. Mothers of selected noncancerous persons	Urban American	107	67.52± .83	12.69± .59	18.79[22]

TABLE 17—*Continued*

B. Mothers—Continued

40	Pearl,[23] Table IV..	Family History Records. Mothers of selected dead nontuberculous persons	Urban American	424	64.72± .52	15.96± .37	24.66 [22]
41	Beeton, Yule, and Pearson,[28] Table IV	Quaker genealogies. Mothers weighted with their fertility..	British	6020 [34]	63.84	.....	.....
42	Wilson and Doering,[37] p. 428	Peirce Genealogy. Mothers with consort's age at death known	American	135	63.0 ±1.7	.....	.....
43	Gould,[25] Fig. 1 ...	Genealogical material. "Mothers not grandmothers"	American	? (ca. 50)	62.96±1.77	.....	.....
44	Wilson and Doering,[37] p. 428	Peirce Genealogy. All mothers	American	188	61.7 ±1.4	.....	.....
45	Beeton, Yule and Pearson,[28] Table I	Quaker genealogies. Age at death of mothers correlated with size of family.....	British	1036	61.18	18.85	30.80 [22]
46	Bell,[31] p. 50	Hyde Genealogy	American	671	60.8	.....	.....
47	Beeton, Yule, and Pearson,[28] Table IV	Whitney Genealogy. Mothers weighted with their fertility..	American	5776 [33]	59.92	.....	.....
48	Pearl,[23] Table IV..	Family History Records. Mothers of dead tuberculous persons	Urban American	424	58.87± .60	18.17± .42	30.86 [22]
49	Beeton and Pearson,[20] Table A ...	Quaker genealogies. Mothers of minor sons	British	795 [21]	58.60± .43	18.14± .31	30.97 [22]
50	Beeton and Pearson,[20] Table A ...	Quaker genealogies. Mothers of minor daughters	British	782 [21]	57.66± .45	18.50± .32	32.08 [22]

TABLE 17—*Continued*

B. Mothers—Continued

ITEM NO.	AUTHORITY	NATURE AND SOURCE OF MATERIAL	KIND OF PEOPLE	N	MEAN (*years*)	STANDARD DEVIATION (*years*)	COEFFICIENT OF VARIATION (*per cent*)
51	Pearl, this study, Table 15	Mothers in FHR Series	Urban American	143	55.56±1.04	18.39± .73	33.10±1.46
52	Pearl,[23] Table IV..	Family History Records. Mothers of living tuberculous persons	Urban American	154	54.55± .81	14.94± .57	27.39 [22]
53	Pearl,[23] Table IV..	Family History Records. Mothers of selected living nontuberculous persons	Urban American	154	54.42± .90	16.47± .63	30.26 [22]
54	Beeton, Yule, and Pearson,[28] Table I	Whitney Genealogy. Age at death of mother correlated with size of family	American	1095	53.29	20.46	38.38 [22]
55	Unweighted averages for mothers, including all (and only) cases for which *N*, mean, and S.D. are given.....			611.8	63.37± .46	16.76± .32	26.45± .54
56	Weighted averages for mothers, including all (and only) cases for which *N*, mean, and S.D. are given.....			10,401 [35]	64.18± .47	17.14± .33	26.71± .55

TABLE 17—*Continued*

C. Grandfathers

57	Pearl, this study, Table 15	Paternal grandfathers of nonagenarians and centenarians...	Chiefly American	365	76.75± .56	15.73± .39	20.49± .53
58	Pearl, this study,	All grandfathers of nonagenarians and centenarians [36]	Chiefly American	730	76.25± .39	15.69± .28	20.58± .38
59	Pearl, this study, Table 15	Maternal grandfathers of nonagenarians and centenarians...	Chiefly American	365	75.74± .55	15.64± .39	20.65± .54
60	Gould,[25] Fig. 1 ..	Genealogical material. Grandfathers	American	1157	70.98± .25	.....	.....
61	Wilson and Doering,[37] p. 429	Peirce Genealogy. Grandfathers weighted with number of sons	American	54	70.5	.....	.....
62	Wilson and Doering,[37] p. 429	Peirce Genealogy. Grandfathers unweighted	American	54	69.5	.....	.....
63	Pearl, this study, Table 15	Paternal grandfathers in FHR Series	Urban American	143	68.99± .92	16.35± .65	23.70±1.00
64	Pearl, this study,	All grandfathers in FHR Series [36]	Urban American	286	68.57± .65	16.38± .47	23.89± .72
65	Pearl, this study, Table 15	Maternal grandfathers in FHR Series	Urban American	143	68.15± .93	16.40± .65	24.07±1.01

TABLE 17—*Continued*

D. Grandmothers

ITEM NO.	AUTHORITY	NATURE AND SOURCE OF MATERIAL	KIND OF PEOPLE	N	MEAN (*years*)	STANDARD DEVIATION (*years*)	COEFFICIENT OF VARIATION (*per cent*)
66	Pearl, this study, Table 15	Paternal grandmothers of nonagenarians and centenarians..	Chiefly American	365	77.05± .56	15.87± .40	20.60± .54
67	Pearl, this study,	All grandmothers of nonagenarians and centenarians [36]	Chiefly American	730	76.78± .39	15.65± .28	20.39± .37
68	Pearl, this study, Table 15	Maternal grandmothers of nonagenarians and centenarians...	Chiefly American	365	76.51± .54	15.43± .39	20.16± .52
69	Gould,[25] Fig. 1...	Genealogical material. Grandmothers	American	872	70.33± .32	.....	.....
70	Pearl, this study, Table 15	Paternal grandmothers in FHR Series	Urban American	143	70.10± .93	16.55± .66	23.61± .99
71	Pearl, this study,	All grandmothers in FHR Series [36]	Urban American	286	7± .67	16.8	± .74
72	Pearl, this study, Table 15	Maternal grandmothers in FHR Series	Urban American	143	67.03± .96	17.08± .68	25.48±1.07

[25] Boston, M., and Karl Pearson. On the inheritance of the duration of life, and on the intensity of natural selection in man. *Biometrika*,

the number of *pairs* of entries in cor-

[20] Beeton, M., and Karl Pearson. On the inheritance of the duration of life, and on the intensity of natural selection in man. *Biometrika*, Vol. 1, pp. 50-89, 1901.

[21] These numbers apparently do not represent the actual numbers of different ancestors. They are the number of *pairs* of entries in correlation tables. But since each ancestor (father or mother) was presumably entered once for each son (or daughter) the actual number of *different* fathers (or mothers) involved is much smaller than the *N* given. In the original paper there is nowhere a clear statement to be found as to exactly how many different ancestors are involved. If they are weighted with fertility the biometric constants for ancestors (fathers and mothers) cannot be precisely or critically interpreted. They do not mean the same thing as do the corresponding constants calculated in the usual way, where each individual is counted but once in computing the mean and standard deviation of the sample. Personally the present authors are unable to regard the means, standard deviations, and coefficients of variation for ... fathers or mothers, as apparently computed in the studies by Pearson and his co-workers, as having ... a *biological* mean... They appear to us to be merely numerical by-products of the computations of correlation coefficients. They may or may not differ significantly from the true means, standard deviations, etc. of the samples of fathers and mothers actually used. No one knows; at least no one is told. We include them here merely because we are bringing together in Table 17, as completely as we are able, all the published material bearing on the subject. How different the mean longevity of parents *may be* when computed with and without fertility weighting is seen in comparing item 26 (unweighted) with item 22 (weighted), item 19 (unweighted) with item 16 (weighted), item 54 (unweighted) with item 47 (weighted), and item 45 (unweighted) with item 41 (weighted) in Table 17.

[22] Computed from original mean and standard deviation by present authors, using all decimals tabulated in original.

[23] Pearl, R. The age at death of the parents of the tuberculous and the cancerous. *Amer. Jour. Hyg.*, Vol. 3, pp. 71-89, 1923.

[24] Pearl, R. Preliminary account of an investigation of factors influencing longevity. *Jour. Amer. Med. Assoc.*, Vol. 82, pp. 259-264, 1924.

[25] Gould, Alice. The longevity of grandparents. *Eug. News*, Vol. 12, pp. 166-171; 175-178, 1927.

[26] Stoessiger, Brenda. On the inheritance of duration of life and cause of death. *Ann. Eug.*, Vol. 5, pp. 105-178, 1933.

[27] Beeton, M., and K. Pearson. Data for the problem of evolution in man. II. A first study of the inheritance of longevity and the selective death-rate in man. *Proc. Roy. Soc.*, Vol. 65, pp. 290-305, 1899.

[28] Beeton, M., G. U. Yule, and K. Pearson. Data for the problem of evolution in man. V. On the correlation between duration of life and the number of offspring. *Proc. Roy. Soc.*, Vol. 67, pp. 159-179, 1900.

[29] This is the total number of father-offspring pairs. The actual number of fathers was 1084, as given in item 24. Cf. footnote 21.

[30] This is the total number of father-offspring pairs. The actual number of fathers was 1000, as given in item 17. Cf. footnote 21.

[31] Bell, A. G. The Duration of Life and Conditions associated with Longevity. A Study of the Hyde Genealogy. *Washington* (Genealogical Record Office) 1918. Pp. 57.

[32] This is the sum of the recorded *N*'s of the samples averaged. But in computing the probable errors the average *N* (683.5) was used. See also footnote 21. The use of the mean *N* for the probable errors is conservative, but considering the heterogeneous character of the groups averaged the resulting probable errors certainly do not overstate our confidence in the combined results.

[33] This is the total number of mother-offspring pairs. The actual number of mothers was 1095, as given in item 50. Cf. footnote 21.

[34] This is the total number of mother-offspring pairs. The actual number of mothers was 1036, as given in item 41. Cf. footnote 21.

[35] This is the sum of the recorded *N*'s of the samples averaged. But in computing the probable errors the average *N* (611.8) was used. See also footnotes 21 and 32.

[36] Constants computed from combined frequency distributions.

[37] Wilson, Edwin B., and C. R. Doering. The elder Peirces. *Proc. Nat. Acad. Sci.*, Vol. 12, pp. 424-432, 1926.

2. Mothers appear to be more variable individually in age at death than fathers, the difference in the weighted mean coefficients of variation being 4.28 ± .70 per cent, a difference large enough to be regarded as safely beyond fluctuations of sampling. Also mothers show a greater range in mean age at death than fathers, the extremes being 53.29 years and 73.33 years, giving a range of 20.04 years. Just as for fathers and grandfathers, mothers appear to have a much greater range in mean ages at death than grandmothers, though here again the available data are too meager to permit any definite conclusions. But for what little it is worth, the range for grandmothers in Table 17 is 10.02 years.

3. Both the unweighted and the weighted mean ages for sections A and B of Table 17 give a higher mean age at death for fathers than for mothers. The differences are small, so small in fact as to be statistically without significance. But the difference, if taken at its face value, contradicts one of the most firmly grounded generalizations of biostatistics, namely that women are generally longer-lived than men. And in so doing it reflects on either (a) the reliability (for the purpose to which they have been put) of the basic materials of the studies from the results of which Table 17 has been built up, or (b) the adequacy and validity of the biometric methods used in the analysis of those materials, or (c) some combination of both (a) and (b). In an earlier paper Pearl [8] has presented evidence warranting grave doubt as to (b) insofar as relates to the correlational technique, and various writers have presented criticisms along the line of (a). Further evidence for scepticism as to the adequacy of conventional biometric methods in the premises will appear as we proceed in the present study.

As has been said Table 17 presents the pertinent available evidence regarding the ages at death of parents and grandparents that has been derived by the application of conventional biometric methods to the data. But besides this there is a certain amount of material pertinent to the problem derived by the application of actuarial methods to essentially the same *kind* of raw data. This material will now be presented in Table 18. To this table is also added an extensive compilation of life table data regarding the average duration of life of male and female human beings generally, which it is hoped will be found useful in various connections.

Wherever possible three columns of numerical data are presented in Table 18, namely $\mathring{e}_{15}+15$, $\mathring{e}_{20}+20$, and $\mathring{e}_{30}+30$. That is, the expectation of life (mean after lifetime) at ages 15, 20 and 30 years, plus the

15, 20, and 30 years respectively lived up to the times of stated expectations. The reason for the choice of these years is that persons who achieve the status of parents first do so, in the vast majority of cases, somewhere between the ages of 15 and 30 years. They have, as individuals, certainly lived that long. The primary purpose of including the figures from general population life tables is to help towards the formation of a judgment on an important question. Do parents (or grandparents) live longer by virtue of (or in association with) their parenthood than do actuarially comparable people in general, that is, people who have lived at least as long as the ages at which persons achieve the status of parenthood?

Inasmuch as a great range of life table data is presented in Table 18, derived from a variety of different kinds of observational material, and since the meaning of the actuarial statistic "expectation of life" ($\mathring{e}_x$) differs somewhat according to the material upon which the life table is based from which it is derived, it seems advisable to make a few precautionary explanations. In Table 18 are included data from life tables computed from five more or less different kinds of raw material, viz. (1) genealogies where only the dead are used, (2) genealogies where both living and dead are used, (3) family history records got by personal interviews with a considerable fraction of the persons involved, where both dead and living are used, (4) records of births, deaths, and total populations in a general population, and (5) the experience of life insurance companies with their policy holders.

Now in the case of (1) each individual is followed throughout his life, and has complete opportunity to live his life out. The expectation of life computed from such material is the actual average number of years lived by the persons involved, after the stated age. In consequence, with such material, expectation of life at birth and mean age at death are identical for the same population.

Under case (4) the expectation of life has not this same direct and clear meaning, because when life tables are constructed from the census (population) and registration (births and deaths) data, the age specific life table death rates (q_x), from which all other life table functions are derived, appertain solely to the forces of natality and mortality for the single year (or short period of years) for which the table is calculated. But in calculating the derived constants, and particularly the expectation of life, the assumption is tacitly made that these particular specific deaths operate (at the respective ages) throughout the life times of the indi-

viduals involved in the table. Now this may or may not be the fact; or, better stated, the actuality may deviate from this postulated state of affairs in a variety of directions and degrees. Insofar as it does so deviate expectation of life at birth will tend to deviate from the mean age at death of the persons actually involved in the table.

Cases (2) and (3) will fall in an intermediate position between (1) and (4), the expectation of life being in any case a less theoretical sort of figure than in (4), and approaching more and more to the precise meaning it has in (1) as the living persons included rise in age. It is actually a weighted average of the ages at death of the dead, and the expectations of life of those living at the time of record.

Life tables constructed from the experience of life insurance companies (5) fall essentially in the same position as (2) and (3). They differ theoretically only because of two complicating factors; namely variable age at entrance into the experience, instead of uniform entrance at birth as in (2) and (3), and lapses of policies (withdrawal from the experience from causes other than death). In a large enough experience the effect of these complicating factors is not serious, or to put it more precisely, may be adequately corrected.

With so much by way of explanation we may proceed to Table 18. In each section of this table the items are arranged in descending order of the values of $\mathring{e}_{20}+20$.

In discussing the data assembled in Table 18 emphasis must first be laid upon the fact that the life table material for parents and grandparents available for citation in sections A, B, C, and D is extremely small, and relates only to American material plus one single Chinese family. It would be foolish to suppose that the means (Items 11, 22, 26, and 30) of the sections mentioned are reliable for purposes of generalization. There is great need for further work along actuarial lines on the problem of the inheritance of life duration, and particularly for the study of material from different racial, social, and economic groups.

Understanding the purely tentative nature of any conclusions, it is of interest to see that the average CML (20) and CML (30)[50] for *fathers* (Item 11) are respectively 3.69 and 2.32 years *greater* than the

[50] For convenience of discussion in the text we have taken CML (20), meaning "computed average longevity" (total duration of life) of a class of persons all living to age 20 before experiencing any mortality, as a shorthand notation, easily set in type, for $\mathring{e}_{20}+20$. Similarly CML (30) means $\mathring{e}_{30}+30$, and CML (15) means $\mathring{e}_{15}+15$.

TABLE 18

Life Table Data on Duration of Life of (a) ancestors, and (b) General and Special Populations

A. Fathers

ITEM NO.	SOURCE	NATURE OF MATERIAL	$\mathring{e}_{15}+15$ (*years*)	$\mathring{e}_{20}+20$ (*years*)	$\mathring{e}_{30}+30$ (*years*)
1	Pearl,[3] p. 263	FHR data. Fathers of persons dying at 80 and over	...	78.5	79.8[38]
2	Pearl,[3] p. 257	FHR data. Fathers of persons dying at 50 and over	...	70.4	70.8[38]
3	Pearl,[3] p. 263	FHR data. Fathers of persons dying under 5	...	66.3	67.5[38]
4	Pearl,[3] p. 257	FHR data. Fathers of persons dying under 50	...	66.1	67.2[38]
5	Yuan,[37a] Table 8	Genealogy of a southern Chinese family. Fathers of sons dying 70 and over years of age	...	62.7	63.9
6	Gould,[25] p. 176	Genealogical data. Parents not grandparents, born 1775-1824	...	...	63.2
7	Yuan,[37a] Table 8	Genealogy of a southern Chinese family. Fathers of sons dying 50-69 years of age	...	60.9	62.2
8	Pearl,[9] Table XXX	Genealogical data	...	...	71.1
9	Yuan,[37a] Table 5	Genealogy of a southern Chinese family. All fathers of sons living 20 or more years	...	59.6	61.0
10	Yuan,[37a] Table 8	Genealogy of a southern Chinese family. Fathers of sons dying 20-40 years of age	...	59.0	60.4
11	Unweighted means	Fathers	...	65.44	66.71

TABLE 18—*Continued*
B. Mothers

ITEM NO.	SOURCE	NATURE OF MATERIAL	$\dot{e}_{15}+15$ (*years*)	$\dot{e}_{20}+20$ (*years*)	$\dot{e}_{30}+30$ (*years*)
12	Pearl,[3] p. 264	FHR data. Mothers of persons dying at 80 and over	...	82.6	82.6 [38]
13	Pearl,[3] p. 258	FHR data. Mothers of persons dying at 50 and over	...	71.6	74.6 [38]
14	Pearl,[3] p. 264	FHR data. Mothers of persons dying under 5	...	69.1	71.3 [38]
15	Pearl,[3] p. 258	FHR data. Mothers of persons dying under 50	...	68.3	70.3 [38]
16	Pearl,[9] Table XXXI	Genealogical data	...	...	69.9
17	Yuan,[37a] Table 8	Genealogy of a southern Chinese family. Mothers of sons dying 70 and over years of age	...	64.6	66.6
18	Gould,[25] p. 176	Genealogical data. Parents not grandparents, born 1775-1824	...	63.7	67.2
19	Yuan,[37a] Table 8	Genealogy of a southern Chinese family. Mothers of sons dying 50-69 years of age	...	63.6	65.2
20	Yuan,[37a] Table 5	Genealogy of a southern Chinese family. All mothers of sons living 20 or more years	...	61.3	63.3
21	Yuan,[37a] Table 8	Genealogy of a southern Chinese family. Mothers of sons dying 20-49 years of age	...	60.5	62.3
22	Unweighted means	Mothers	...	67.26	69.33

TABLE 18—*Continued*

C. Grandfathers

ITEM NO.	SOURCE	NATURE OF MATERIAL	$\mathring{e}_{15}+15$ (*years*)	$\mathring{e}_{20}+20$ (*years*)	$\mathring{e}_{30}+30$ (*years*)
23	Pearl,[3] p. 259	FHR data. Grandfathers of persons dying at 50 and over	...	70.6	72.2 [38]
24	Gould,[25] p. 176	Genealogical data. Grandfathers born 1775-1824	...	...	71.6
25	Pearl,[3] p. 259	FHR data. Grandfathers of persons dying under 50	...	67.2	67.9 [38]
26	Unweighted means	Grandfathers	...	68.90	70.57

D. Grandmothers

27	Pearl,[3] p. 260	FHR data. Grandmothers of persons dying at 50 and over	...	77.4	79.9 [38]
28	Gould,[25] p. 176	Genealogical data. Grandmothers born 1775-1824	...	70.8	71.3
29	Pearl,[3] p. 260	FHR data. Grandmothers of persons dying under 50	...	68.8	70.1 [38]
30	Unweighted means	Grandmothers	...	72.33	73.77

TABLE 18—*Continued*

E. General and Special Populations

I. Males

ITEM NO.	SOURCE	NATURE OF MATERIAL	$\mathring{e}_{15}+15$ (*years*)	$\mathring{e}_{20}+20$ (*years*)	$\mathring{e}_{30}+30$ (*years*)
31	Inst. Int. de Statistique,[39] p. 446	Denmark, 1921-1925	....	69.40	70.80
32	Inst. Int. de Statistique,[39] p. 449	New Zealand, 1921-1922	....	68.66	69.98
33	Inst. Int. de Statistique,[39] p. 448	Sweden, 1921-1925	....	68.49	70.66
34	Inst. Int. de Statistique,[39] p. 449	New Zealand, 1911-1915	....	67.61	69.03
35	Inst. Int. de Statistique,[39] p. 446	Saxony, 1924-1926	....	67.43	69.02
36	Inst. Int. de Statistique,[39] p. 449	New Zealand, 1906-1910	....	67.20	68.78
37	Inst. Int. de Statistique,[39] p. 449	Australia, 1920-1922	....	66.99	68.44
38	Inst. Int. de Statistique,[39] p. 449	New Zealand, 1901-1905	....	66.74	68.47
39	Inst. Int. de Statistique,[39] p. 446	Wurtemberg, 1924-1926	....	66.72	68.68
40	Inst. Int. de Statistique,[39] p. 447	Holland, 1910-1920	....	66.70	68.80
41	Inst. Int. de Statistique,[39] p. 446	Germany, 1924-1926	....	66.70	68.56
42	Inst. Int. de Statistique,[39] p. 446	Denmark, 1911-1915	....	66.70	68.40
43	Inst. Int. de Statistique,[39] p. 446	Bavaria, 1924-1926	....	66.60	68.13
44	DePorte,[40] p. 306	New York State, 1910. Native whites of native parentage	....	66.57	68.31
45	Inst. Int. de Statistique,[39] p. 449	New Zealand, 1896-1900	....	66.34	67.19
46	Glover[7] p. 216	Denmark, 1906-1910	65.5	66.3	68.0
47	Inst. Int. de Statistique,[39] p. 449	Union of South Africa, 1925-1927 Europeans	....	66.27	67.87
48	Inst. Int. de Statistique,[39] p. 448	White Russia, 1926-1927	....	66.22	68.58
49	Bull. Met. Life Ins. Co.,[45] p. 8	American physicians. White. 1925	....	....	68.01
50	Inst. Int. de Statistique,[39] p. 446	Denmark, 1916-1920	....	66.10	68.90
51	Glover,[7] p. 112	U. S. A. Original Registration States. 1901. Whites in rural parts	65.01	65.97	68.37

	Whites in rural parts	65.01	65.97	68.37
52 Glover,[7] p. 232	American-Canadian Mortality Investigation, 1900-1915	65.06	65.93	67.70
53 Glover,[7] p. 114	U. S. A. Original Registration States. 1910. Whites in rural parts	65.10	65.92	68.10
54 Glover,[7] p. 216	Sweden, 1901-1910	64.79	65.88	68.57
55 Inst. Int. de Statistique,[39] p. 447	England and Wales, 1920-1922	....	65.78	67.40
56 Glover,[7] p. 216	Holland, 1900-1909	64.8	65.7	67.8
57 Inst. Int. de Statistique,[39] p. 448	North Ireland, 1925-1927	....	65.63	67.46
58 Glover,[7] p. 232	Seventeen Swedish Offices Life Tables, 1895-1906	64.01	65.59	66.99
59 Inst. Int. de Statistique,[39] p. 449	New Zealand, 1891-1895	....	65.47	67.54
60 Dublin, Kopf, and Lotka,[46] p. 330	U. S. Registration Area. 1922. Whites	64.59	65.42	67.31
61 Inst. Int. de Statistique,[39] p. 446	Denmark, 1901-1905	....	65.40	67.40
62 Inst. Int. de Statistique,[39] p. 448	Sweden, 1911-1920	....	65.27	68.57
63 Inst. Int. de Statistique,[39] p. 449	Union of South Africa, 1920-1922. Europeans	....	65.26	67.08
64 Inst. Int. de Statistique,[39] p. 447	Italy, 1921-1922	....	65.17	67.67
65 Glover,[7] p. 216	Norway, 1901-1910	63.69	65.16	68.86
66 Bull. Met. Life Ins. Co.,[44] p. 7	U. S. Registration Area. 1919-1920. Whites	64.04	64.94	67.14
67 Inst. Int. de Statistique,[39] p. 449	Siberia (U. R. S. S.), 1926-1927	....	64.86	67.29
68 Inst. Int. de Statistique,[39] p. 447	Norway, 1911-1920	....	64.85	68.83
69 Dublin,[40] p. 96	Russian born whites (mostly Jews) living in New York State, 1910	....	64.84	....
70 Inst. Int. de Statistique,[39] p. 447	Scotland, 1921	....	64.82	66.52
71 Inst. Int. de Statistique,[39] p. 448	Sweden, 1891-1900	....	64.75	67.50
72 Glover,[7] p. 216	Australia, 1901-1910	64.03	64.74	66.52
73 Inst. Int. de Statistique,[39] p. 448	Ukrainia, 1926-1927	....	64.58	67.25
74 Gould,[25] p. 176	American genealogical population, born 1775-1824	63.81	64.53	67.83
75 Inst. Int. de Statistique,[39] p. 446	Denmark, 1895-1900	....	64.50	66.80

TABLE 18—*Continued*

E. General and Special Populations (Continued)

I. Males (Continued)

ITEM NO.	SOURCE	NATURE OF MATERIAL	$\mathring{e}_{15}+15$ (*years*)	$\mathring{e}_{20}+20$ (*years*)	$\mathring{e}_{30}+30$ (*years*)
76	Inst. Int. de Statistique,[39] p. 448	Ukrainia, 1925-1926	....	64.49	67.18
77	Dublin,[49] p. 96	Italian born whites, living in New York State, 1910	....	64.26	....
78	Inst. Int. de Statistique,[39] p. 447	England and Wales, 1910-1912	....	64.21	65.81
79	Inst. Int. de Statistique,[39] p. 447	Italy, 1910-1912	....	64.20	66.73
80	Pearl,[9] p. 235	FHR data. Moderate drinkers	....	....	66.75
81	Pearl,[9] p. 235	FHR data. Abstainers	....	....	66.34
82	Inst. Int. de Statistique,[39] p. 448	Sweden, 1881-1890	....	64.18	66.87
83	Jones,[42] p. 29	Glasgow, 1909-1912. Four room houses and up	63.54	64.16	....
84	Inst. Int. de Statistique,[39] p. 446	Saxony, 1900/01-1910/11	....	64.14	66.02
85	Bull. Met. Life Ins. Co.,[43] p. 1	Industrial policy holders. 1921. Whites	63.28	64.01	65.74
86	Inst. Int. de Statistique,[39] p. 447	Norway. 1881/82-1890/91	....	63.89	67.68
87	Inst. Int. de Statistique,[39] p. 448	Switzerland, 1920-1921	....	63.85	65.56
88	Jones,[42] p. 29	Glasgow, 1909-1912. Three room houses	63.06	63.81	....
89	Inst. Int. de Statistique,[39] p. 446	Denmark, 1885-1894	....	63.60	66.20
90	Inst. Int. de Statistique,[39] p. 447	Norway, 1891/92-1900/01	....	63.58	67.69
91	Inst. Int. de Statistique,[39] p. 446	Prussia, 1911-1914	....	63.46	65.40
92	Inst. Int. de Statistique,[39] p. 446	Germany, 1910-1911	....	63.43	65.29
93	Inst. Int. de Statistique,[39] p. 447	Holland, 1890-1899	....	63.40	65.90
94	Bull. Met. Life Ins. Co.,[43] p. 1	Industrial policy holders. 1930. Whites	62.79	63.35	64.75
95	Glover,[7] p. 90	U. S. A. Original Registration States. 1910. Native whites	62.52	63.32	65.61
96	Glover,[7] p. 216	Italy, 1901-1910	62.17	63.27	65.94

		Native whites	62.52	63.32	65.61
96	Glover,[7] p. 216	Italy, 1901-1910	62.17	63.27	65.94
97	Inst. Int. de Statistique,[39] p. 447	Scotland, 1911	....	63.27	65.17
98	Glover,[7] p. 88	U. S. A. Original Registration States. 1901. Native whites	62.28	63.24	66.08
99	Inst. Int. de Statistique,[39] p. 448	U. R. S. S. European Russia, 1926-1927	....	63.24	65.65
100	Inst. Int. de Statistique,[39] p. 446	Denmark, 1880-1889	....	63.20	65.90
101	Inst. Int. de Statistique,[39] p. 447	Norway, 1856-1865	....	63.16	66.28
102	Inst. Int. de Statistique,[39] p. 447	Norway, 1871/72-1880/81	....	63.14	66.82
103	Inst. Int. de Statistique,[39] p. 446	Bavaria, 1911-1913	....	63.11	64.93
104	Inst. Int. de Statistique,[39] p. 447	Italy, 1899-1902	....	63.10	65.65
105	Glover,[7] p. 232	Thirty American Offices Life Tables, 1874	61.57	63.07	65.85
106	Glover,[7] p. 216	England, 1901-1910	62.31	63.01	64.76
107	Inst. Int. de Statistique,[39] p. 447	France, 1920-1923	....	62.93	65.50
108	Inst. Int. de Statistique,[39] p. 446	Wurtemberg, 1901-1910	....	62.93	64.94
109	Inst. Int. de Statistique,[39] p. 448	Ukrainia, 1924	....	62.84	....
110	Inst. Int. de Statistique,[39] p. 449	Australia, 1891-1900	....	62.81	65.11
111	Inst. Int. de Statistique,[39] p. 446	Prussia, 1906-1910	....	62.81	64.75
112	Glover,[7] p. 68	U. S. A. Original Registration States, 1910. Whites	61.91	62.71	64.87
113	Glover,[7] p. 216	Germany, 1901-1910	61.71	62.56	64.55
114	Inst. Int. de Statistique,[39] p. 448	Russian Soc. Fed. Sov. Rep., 1926-1927	....	62.48	64.82
115	Glover,[7] p. 58	U. S. A. Original Registration States, 1910	61.66	62.48	64.70
116	Glover,[7] p. 232	British Offices Life Tables. 1863-1893. $O^{M(5)}$	60.98	62.39	65.06
117	Glover,[7] p. 66	U. S. A. Original Registration States, 1901-1910. Whites	61.50	62.39	64.80
118	Inst. Int. de Statistique,[39] p. 447	Holland, 1880-1889	....	62.30	65.10
119	Inst. Int. de Statistique,[39] p. 448	Sweden, 1871-1880	....	62.30	65.10
120	Inst. Int. de Statistique,[39] p. 448	White Russia, 1896-1897	....	62.23	64.82
121	Dublin,[49] p. 96	English, Scotch and Welsh born whites, living in New York State, 1910	....	62.23	....
122	Glover,[7] p. 232	American Experience Mortality Tables, 1860	60.50	62.20	65.33

TABLE 18—*Continued*
E. General and Special Populations (Continued)
I. Males (Continued)

ITEM NO.	SOURCE	NATURE OF MATERIAL	$\mathring{e}_{15}+15$ (*years*)	$\mathring{e}_{20}+20$ (*years*)	$\mathring{e}_{30}+30$ (*years*)
123	Jones,[42] p. 41	Glasgow, 1920-1922	61.38	62.20	63.92
124	Glover,[7] p. 64	U. S. A. Original Registration States, 1901. Whites	61.25	62.19	64.88
125	Inst. Int. de Statistique,[39] p. 447	Finland, 1901-1910	....	62.16	64.94
126	Glover,[7] p. 56	U. S. A. Original Registration States, 1901	61.06	62.03	64.76
127	Inst. Int. de Statistique,[39] p. 446	Bavaria, 1901-1910	....	61.93	64.01
128	Inst. Int. de Statistique,[39] p. 446	Belgium, 1891-1900	....	61.80	64.20
129	Glover,[7] p. 98	U. S. A. Original Registration States, 1910. Foreign-born whites	60.89	61.75	63.71
130	Pearl,[9] p. 235	FHR data. All drinkers	....	....	63.60
131	Glover,[7] p. 216	Switzerland, 1901-1910	60.86	61.70	63.80
132	Inst. Int. de Statistique,[39] p. 448	Ukrainia, 1895-1898	....	61.70	....
133	Inst. Int. de Statistique,[39] p. 446	Wurtemberg, 1891-1900	....	61.68	63.95
134	Inst. Int. de Statistique,[39] p. 446	Denmark, 1860-1869	....	61.60	64.50
135	Glover,[7] p. 216	France, 1898-1903	60.45	61.53	64.35
136	Inst. Int. de Statistique,[39] p. 446	Prussia, 1900-1901	....	61.53	63.69
137	Inst. Int. de Statistique,[39] p. 447	Iceland, 1901-1910	....	61.50	65.40
138	Inst. Int. de Statistique,[39] p. 447	France, 1908-1913	....	61.39	64.03
139	Inst. Int. de Statistique,[39] p. 446	Prussia, 1891-1900	....	61.30	63.52
140	Inst. Int. de Statistique,[39] p. 446	Belgium, 1881-1890	....	61.29	64.06
141	DePorte,[40] p. 310	New York State, 1910. Foreign-born whites	....	61.26	63.08
142	Inst. Int. de Statistique,[39] p. 446	Germany, 1891-1900	....	61.23	63.46
143	Inst. Int. de Statistique,[39] p. 447	France, 1861-1865	....	61.20	64.65
144	Jones,[42] p. 29	Glasgow, 1909-1912. Two room houses	60.33	61.19	....

144	Jones,[42] p. 29	Glasgow, 1909-1912. Two room houses	60.33	61.19	....
145	Inst. Int. de Statistique,[39] p. 446	Saxony, 1891-1900	....	61.16	63.08
146	Dublin and Whitney,[48] p. 442	Industrial policy holders. 1911-1916. Whites. Tuberculosis absent	60.5	61.1	62.6
147	Inst. Int. de Statistique,[39] p. 447	England and Wales, 1891-1900	....	61.02	63.07
148	Inst. Int. de Statistique,[39] p. 448	Sweden, 1861-1870	....	61.00	63.60
149	Inst. Int. de Statistique,[39] p. 448	U. R. S. S. European Russia, 1896-1897	....	60.98	63.78
150	Inst. Int. de Statistique,[39] p. 446	Austria, 1906-1910	....	60.90	63.49
151	Inst. Int. de Statistique,[39] p. 446	Bavaria, 1891-1900	....	60.80	63.13
152	Dublin,[49] p. 96	German born whites, living in New York State, 1910	....	60.80	....
153	Glover,[7] p. 96	U. S. A. Original Registration States. 1901. Foreign-born whites	59.71	60.68	63.07
154	Inst. Int. de Statistique,[39] p. 448	Russian Soc. Fed. Sov. Rep., 1896-1897	....	60.60	63.37
155	Inst. Int. de Statistique,[39] p. 449	Australia, 1881-1890	....	60.58	63.64
156	Glover,[7] p. 106	U. S. A. Original Registration States, 1910. Whites in cities	59.72	60.51	62.61
157	Inst. Int. de Statistique,[39] p. 446	Austria, 1901-1905	....	60.50	63.27
158	Inst. Int. de Statistique,[39] p. 448	Switzerland, 1889-1900	....	60.50	62.90
159	Inst. Int. de Statistique,[39] p. 447	France, 1877-1881	....	60.42	63.83
160	Inst. Int. de Statistique,[39] p. 447	Scotland, 1891-1900	....	60.36	62.95
161	Inst. Int. de Statistique,[39] p. 447	Italy, 1876-1887	....	60.35	63.50
162	Glover,[7] p. 216	Japan, 1898-1903	59.02	60.35	63.44
163	Inst. Int. de Statistique,[39] p. 447	Holland, 1870-1879	....	60.30	63.70
164	Inst. Int. de Statistique,[39] p. 447	England and Wales, 1881-1890	....	60.27	62.52
165	Inst. Int. de Statistique,[39] p. 447	Scotland, 1881-1890	....	60.19	63.06
166	Inst. Int. de Statistique,[39] p. 446	Austria, 1895-1900	....	60.17	63.03
167	Inst. Int. de Statistique,[39] p. 446	Prussia, 1890-1891	....	60.07	62.50
168	Inst. Int. de Statistique,[39] p. 447	France, 1817-1831	....	60.00	64.00
169	Inst. Int. de Statistique,[39] p. 446	Wurtemberg, 1876-1880	....	60.00	62.80

TABLE 18—*Continued*

E. General and Special Populations (Continued)

I. Males (Continued)

ITEM NO.	SOURCE	NATURE OF MATERIAL	$\mathring{e}_{15}+15$ (*years*)	$\mathring{e}_{20}+20$ (*years*)	$\mathring{e}_{30}+30$ (*years*)
170	Inst. Int. de Statistique,[39] p. 448	Sweden, 1856-1860	....	59.99	62.91
171	Inst. Int. de Statistique,[39] p. 446	Denmark, 1835-1844	....	59.80	62.60
172	Inst. Int. de Statistique,[39] p. 448	Switzerland, 1881-1888	....	59.58	62.21
173	Inst. Int. de Statistique,[39] p. 446	Germany, 1881-1890	....	59.52	62.11
174	Inst. Int. de Statistique,[39] p. 447	England and Wales, 1838-1854	....	59.48	62.76
175	Inst. Int. de Statistique,[39] p. 447	England and Wales, 1871-1880	....	59.40	62.10
176	Bull. Met. Life Ins. Co.,[43] p. 1	Industrial policy holders, 1921. Colored	58.06	59.24	62.17
177	DePorte,[40] p. 308	New York State. 1910. Native white of foreign or mixed parentage	....	59.20	61.93
178	Glover,[7] p. 104	U. S. A. Original Registration States. 1901. Whites in cities	58.15	59.13	61.89
179	Inst. Int. de Statistique,[39] p. 449	Japan, 1921-1925	....	59.10	62.59
180	Jones,[42] p. 29	Glasgow, 1909-1912. One room house	57.99	58.98	....
181	Inst. Int. de Statistique,[39] p. 448	Sweden, 1841-1845	....	58.95	61.60
182	Inst. Int. de Statistique,[39] p. 447	Scotland, 1861-1870	....	58.75	62.20
183	Inst. Int. de Statistique,[39] p. 448	Switzerland, 1876-1880	....	58.70	61.70
184	Inst. Int. de Statistique,[39] p. 447	Scotland, 1871-1880	....	58.68	62.05
185	Inst. Int. de Statistique,[39] p. 448	Sweden, 1846-1850	....	58.58	61.18
186	Inst. Int. de Statistique,[39] p. 446	Germany, 1871/72-1880/81	....	58.45	61.41
187	Dublin and Whitney,[48] p. 442	Industrial policy holders, 1911-1916. Colored. Tuberculosis absent	57.2	58.2	60.6
188	Glover,[7] p. 232	Three Japanese Offices Life Tables. 1905. $J^{M(5)}$	56.15	58.16	61.88
189	Inst. Int. de Statistique,[39] p. 448	Sweden, 1851-1855	....	58.11	60.89

189	Inst. Int. de Statistique,[39] p. 448	Sweden, 1851-1855	....	58.11	60.89
190	Inst. Int. de Statistique,[39] p. 446	Prussia, 1867-1877	....	58.08	61.18
191	Inst. Int. de Statistique,[39] p. 447	Holland, 1850-1859	....	57.95	61.44
192	Inst. Int. de Statistique,[39] p. 447	Finland, 1911-1920	....	57.91	62.49
193	Yuan,[47] p. 167	Genealogy of a southern Chinese family. All entries	....	57.7	59.6
194	Inst. Int. de Statistique,[39] p. 447	Iceland, 1890-1901	....	57.67	61.66
195	Glover,[7] p. 232	American Standard Industrial Mortality Table. 1896-1905	56.55	57.50	60.62
196	Dublin and Whitney,[48] p. 442	Industrial policy holders, 1911-1916. Whites. Tuberculosis present	56.7	57.4	59.8
197	Inst. Int. de Statistique,[39] p. 448	Sweden, 1816-1840	....	57.32	60.25
198	Bull. Met. Life Ins. Co.,[43] p. 1	Industrial policy holders. 1911-1912. Whites	56.12	56.87	59.36
199	Inst. Int. de Statistique,[39] p. 446	Austria, 1870-1880	....	56.80	60.53
200	Yuan,[47] p. 168	Genealogy of a southern Chinese family. Births between 1700 and 1749	....	56.6	60.0
201	Bull. Met. Life Ins. Co.,[43] p. 1	Industrial policy holders. 1930. Colored	55.32	56.49	59.55
202	Inst. Int. de Statistique,[39] p. 447	Iceland, 1850-1860	....	56.42	60.63
203	Hernando,[41] p. 174	Manila, 1920. Native born	54.21	55.69	59.11
204	Pearl,[9] p. 235	FHR data. Heavy drinkers	....	....	58.57
205	Glover,[7] p. 76	Original Registration States, 1901. Negroes	53.26	55.11	59.25
206	Yuan,[47] p. 169	Genealogy of a southern Chinese family. Births between 1750 and 1799	....	54.8	57.9
207	Inst. Int. de Statistique,[39] p. 447	Holland, 1840-1851	....	54.62	59.74
208	Bull. Met. Life Ins. Co.,[43] p. 1	Industrial policy holders. 1911-1912. Colored	52.42	54.16	57.95
209	Dublin and Whitney,[48] p. 442	Industrial policy holders. 1911-1916. Colored. Tuberculosis present	52.1	53.8	57.7
210	Glover,[7] p. 78	U. S. A. Original Registration States, 1901-1910. Negroes	51.98	53.78	57.97
211	Yuan,[47] p. 169	Genealogy of a southern Chinese family. Births between 1800-1849	....	53.7	57.4

TABLE 18—*Continued*

E. General and Special Populations (Continued)

I. Males (Continued)

ITEM NO.	SOURCE	NATURE OF MATERIAL	$\dot{e}_{15}+15$ (*years*)	$\dot{e}_{20}+20$ (*years*)	$\dot{e}_{30}+30$ (*years*)
212	Glover,[7] p. 80	U. S. A. Original Registration States, 1910. Negroes	51.77	53.46	57.33
213	Jones,[42] p. 35	Glasgow, 1870-1872	51.57	53.42	56.91
214	Yuan,[47] p. 168	Genealogy of a southern Chinese family. Births between 1600 and 1699	....	52.9	57.9
215	Dublin,[49] p. 96	Irish born whites, living in New York State, 1910	....	51.35	....
216	Glover,[7] p. 216	India, 1901-1910	45.32	47.46	52.44
217	Unweighted means	Males	59.95	61.75	64.39

II. Females

218	Inst. Int. de Statistique,[39] p. 449	New Zealand, 1921-1922	....	70.36	71.76
219	Inst. Int. de Statistique,[39] p. 449	Australia, 1920-1922	....	70.03	71.48
220	Inst. Int. de Statistique,[39] p. 448	Sweden, 1921-1925	....	69.73	71.68
221	Jones,[42] p. 29	Glasgow, 1909-1912. Four room house and up	69.20	69.62	....
222	Inst. Int. de Statistique,[39] p. 449	Union of South Africa, 1925-1927. Europeans	....	69.34	70.77
223	Inst. Int. de Statistique,[39] p. 446	Denmark, 1921-1925	....	69.30	70.80
224	Inst. Int. de Statistique,[39] p. 446	Saxony, 1924-1926	....	69.19	70.73

224	Inst. Int. de Statistique,[39] p. 446	Saxony, 1924-1926	....	69.19	70.73
225	Inst. Int. de Statistique,[39] p. 449	New Zealand, 1911-1915	....	69.14	70.53
226	Inst. Int. de Statistique,[39] p. 449	New Zealand, 1906-1910	....	68.77	70.48
227	Inst. Int. de Statistique,[39] p. 447	England and Wales, 1920-1922	....	68.73	70.26
228	Inst. Int. de Statistique,[39] p. 448	White Russia, 1926-1927	....	68.61	71.79
229	Inst. Int. de Statistique,[39] p. 446	Denmark, 1911-1915	....	68.40	70.20
230	Inst. Int. de Statistique,[39] p. 449	New Zealand, 1901-1905	....	68.23	70.06
231	Glover,[7] p. 218	Denmark, 1906-1910	67.4	68.2	70.1
232	Inst. Int. de Statistique,[39] p. 449	Union of South Africa. 1920-1922. Europeans	....	68.15	69.93
233	Inst. Int. de Statistique,[39] p. 446	Germany, 1924-1926	....	68.09	69.76
234	Inst. Int. de Statistique,[39] p. 449	Siberia (U. R. S. S.), 1926-1927	....	67.98	70.59
235	Inst. Int. de Statistique,[39] p. 449	New Zealand, 1896-1900	....	67.91	69.72
236	Pearl,[9] p. 239	FHR data. Moderate drinkers	....	....	70.95
237	Glover,[7] p. 218	Sweden, 1901-1910	66.48	67.66	70.20
238	Inst. Int. de Statistique,[39] p. 446	Wurtemberg, 1924-1926	....	67.55	69.16
239	Glover,[7] p. 218	Australia, 1901-1910	66.86	67.52	69.33
240	Inst. Int. de Statistique,[39] p. 447	Holland, 1910-1920	....	67.50	69.50
241	Inst. Int. de Statistique,[39] p. 446	Denmark, 1901-1905	....	67.50	66.60
242	Inst. Int. de Statistique,[39] p. 448	Russian Soc. Fed. Sov. Rep., 1926-1927	....	67.48	69.79
243	Inst. Int. de Statistique,[39] p. 448	Sweden, 1911-1920	....	67.37	70.18
244	Inst. Int. de Statistique,[39] p. 448	U. R. S. S. European Russia, 1926-1927	....	67.36	69.75
245	Glover,[7] p. 218	Norway, 1901-1910	66.05	67.35	70.24
246	Inst. Int. de Statistique,[39] p. 446	Denmark, 1916-1920	....	67.20	69.60
247	Inst. Int. de Statistique,[39] p. 447	Norway, 1911-1920	....	67.19	70.35
248	Inst. Int. de Statistique,[39] p. 449	New Zealand, 1891-1895	....	67.19	69.33
249	Inst. Int. de Statistique,[39] p. 446	Bavaria, 1924-1926	....	67.17	68.63
250	Inst. Int. de Statistique,[39] p. 447	England and Wales, 1910-1912	....	67.10	68.54
251	Bull. Met. Life Ins. Co.,[43] p. 1	Industrial policy holders. 1930. Whites	66.49	67.01	68.47
252	Glover,[7] p. 218	Holland, 1900-1909	66.0	66.9	68.8

TABLE 18—*Continued*

E. General and Special Populations (Continued)

II. Females (Continued)

ITEM NO.	SOURCE	NATURE OF MATERIAL	$\mathring{e}_{15}+15$ (*years*)	$\mathring{e}_{20}+20$ (*years*)	$\mathring{e}_{30}+30$ (*years*)
253	Glover,[7] p. 118	U. S. A. Original Registration States, 1910. Whites in rural parts	66.07	66.86	69.05
254	Inst. Int. de Statistique,[39] p. 447	Scotland, 1921	....	66.82	68.63
255	Inst. Int. de Statistique,[39] p. 448	Ukrainia, 1926-1927	....	66.76	69.34
256	Inst. Int. de Statistique,[39] p. 448	Sweden, 1891-1900	....	66.76	69.31
257	Inst. Int. de Statistique,[39] p. 446	Denmark, 1895-1900	....	66.70	68.90
258	Dublin,[40] p. 96	Russian born whites (mostly Jews), living in New York State. 1910	....	66.60	....
259	Inst. Int. de Statistique,[39] p. 447	Norway, 1891/92-1900/01	....	66.54	69.43
260	Inst. Int. de Statistique,[39] p. 447	Iceland, 1901-1910	....	66.50	69.40
261	Dublin, Kopf, and Lotka,[46] p. 332	U. S. Registration Area, 1922. Whites	65.62	66.44	68.42
262	Inst. Int. de Statistique,[39] p. 448	Sweden, 1881-1890	....	66.40	68.82
263	Inst. Int. de Statistique,[39] p. 448	Ukrainia, 1925-1926	....	66.37	69.08
264	Inst. Int. de Statistique,[39] p. 447	France, 1920-1923	....	66.16	68.61
265	Glover,[7] p. 116	U. S. A. Original Registration States, 1901. Whites in rural parts	65.06	66.09	68.80
266	Jones,[42] p. 29	Glasgow, 1909-1912. Three room house	65.32	66.00	....
267	Inst. Int. de Statistique,[39] p. 447	Italy, 1921-1922	....	65.92	68.33
268	Inst. Int. de Statistique,[39] p. 447	Norway, 1881/82-1890/91	....	65.90	68.81
269	Bull. Met. Life Ins. Co.,[43] p. 1	Industrial policy holders, 1921. Whites	65.12	65.88	67.74
270	Inst. Int. de Statistique,[39] p. 448	Switzerland, 1920-1921	....	65.85	67.79
271	Pearl,[9] p. 239	FHR data. Abstainers	....	....	67.63
272	Inst. Int. de Statistique,[39] p. 446	Prussia, 1911-1914	....	65.81	67.72
273	Bull. Met. Life Ins. Co.,[44] p. 8	U. S. Registration Area, 1919-1920. Whites	64.94	65.77	68.25

272	Inst. Int. de Statistique,[39] p. 446	Prussia, 1911-1914	....	65.81	67.72
273	Bull. Met. Life Ins. Co.,[44] p. 8	U. S. Registration Area, 1919-1920. Whites	64.94	65.77	68.25

274	Glover,[7] p. 218	England, 1901-1910	65.08	65.77	67.36
275	Glover,[7] p. 94	U. S. A. Original Registration States, 1910. Native whites	64.98	65.76	67.98
276	Inst. Int. de Statistique,[39] p. 449	Australia, 1891-1900	....	65.72	67.86
277	Dublin,[40] p. 96	German born whites, living in New York State, 1910	....	65.57	....
278	Inst. Int. de Statistique,[39] p. 446	Denmark, 1885-1894	....	65.40	67.90
279	Inst. Int. de Statistique,[39] p. 447	Norway, 1871/72-1880/81	....	65.39	68.19
280	Inst. Int. de Statistique,[39] p. 446	Germany, 1910-1911	....	65.35	67.30
281	Inst. Int. de Statistique,[39] p. 447	Scotland, 1911	....	65.35	67.22
282	Inst. Int. de Statistique,[39] p. 446	Prussia, 1906-1910	....	65.32	67.29
283	Inst. Int. de Statistique,[39] p. 447	Norway, 1856-1865	....	65.27	67.81
284	Inst. Int. de Statistique,[39] p. 448	North Ireland, 1925-1927	....	65.22	67.42
285	Inst. Int. de Statistique,[39] p. 448	Sweden, 1871-1880	....	65.00	67.50
286	Glover,[7] p. 92	U. S. A. Original Registration States. 1901. Native whites	63.96	64.97	67.81
287	Gould,[25] p. 176	American genealogical population, born 1775-1824	64.16	64.96	68.12
288	Dublin,[40] p. 96	Italian born whites, living in New York State, 1910	....	64.94	....
289	Inst. Int. de Statistique,[39] p. 446	Denmark, 1880-1889	....	64.90	67.60
290	Glover,[7] p. 74	U. S. A. Original Registration States, 1910. Whites	64.12	64.88	66.96
291	Glover,[7] p. 218	Germany, 1901-1910	64.00	64.84	66.94
292	Inst. Int. de Statistique,[39] p. 447	France, 1908-1913	....	64.83	67.67
293	Inst. Int. de Statistique,[39] p. 447	Holland, 1890-1899	....	64.80	67.10
294	Dublin and Whitney,[43] p. 442	Industrial policy holders, 1911-1916. Whites. Tuberculosis absent	64.3	64.8	66.1
295	Inst. Int. de Statistique,[39] p. 447	Iceland, 1890-1901	....	64.69	66.89
296	Inst. Int. de Statistique,[39] p. 447	Italy, 1910-1912	....	64.67	67.33

TABLE 18—*Continued*

E. General and Special Populations (Continued)

II. Females (Continued)

ITEM NO.	SOURCE	NATURE OF MATERIAL	$\mathring{e}_{15}+15$ (*years*)	$\mathring{e}_{20}+20$ (*years*)	$\mathring{e}_{30}+30$ (*years*)
297	Glover,[7] p. 62	U. S. A. Original Registration States, 1910	63.87	64.66	66.79
298	Inst. Int. de Statistique,[39] p. 447	Finland, 1901-1910	....	64.54	67.30
299	Jones,[42] p. 44	Glasgow, 1920-1922	63.76	64.53	66.40
300	Inst. Int. de Statistique,[39] p. 446	Bavaria, 1911-1913	....	64.48	66.44
301	Inst. Int. de Statistique,[39] p. 446	Belgium, 1891-1900	....	64.45	66.95
302	Glover,[7] p. 72	U. S. A. Original Registration States, 1901-1910. Whites	63.51	64.39	66.75
303	Inst. Int. de Statistique,[39] p. 446	Prussia, 1900-1901	....	64.27	66.42
304	Inst. Int. de Statistique,[39] p. 446	Wurtemberg, 1901-1910	....	64.23	66.36
305	Inst. Int. de Statistique,[39] p. 448	Ukrainia, 1924	....	64.10	....
306	Glover,[7] p. 218	France, 1898-1903	62.90	64.02	66.93
307	Dublin,[49] p. 96	English, Scotch and Welsh born whites, living in New York State, 1910	....	64.01	....
308	Inst. Int. de Statistique,[39] p. 446	Saxony, 1891-1900	....	63.96	66.23
309	Inst. Int. de Statistique,[39] p. 448	Sweden, 1861-1870	....	63.90	66.20
310	Inst. Int. de Statistique,[39] p. 447	Finland, 1911-1920	....	63.82	66.96
311	Inst. Int. de Statistique,[39] p. 447	France, 1817-1831	....	63.80	63.40
312	Glover,[7] p. 70	U. S. A. Original Registration States, 1901. Whites	62.79	63.77	66.42
313	Inst. Int. de Statistique,[39] p. 446	Prussia, 1891-1900	....	63.76	65.94
314	Inst. Int. de Statistique,[39] p. 446	Belgium, 1881-1890	....	63.74	66.55
315	Glover,[7] p. 218	Italy, 1901-1910	62.43	63.69	66.58
316	Glover,[7] p. 218	Switzerland, 1901-1910	62.62	63.69	66.10

315	Glover,[7] p. 218	Italy, 1901-1910	62.43	63.69	66.58
316	Glover,[7] p. 218	Switzerland, 1901-1910	62.62	63.69	66.10
317	Inst. Int. de Statistique,[39] p. 449	Australia, 1881-1890	....	63.63	66.13
318	Glover,[7] p. 60	U. S. A. Original Registration States, 1901	62.60	63.60	66.30
319	Inst. Int. de Statistique,[39] p. 447	Iceland, 1850-1860	....	63.53	66.28
320	Glover,[7] p. 110	U. S. A. Original Registration States, 1910. Whites in cities	62.77	63.51	65.52
321	Inst. Int. de Statistique,[39] p. 447	Holland, 1880-1889	....	63.50	66.10
322	Glover,[7] p. 102	U. S. A. Original Registration States, 1910. Foreign born whites	62.79	63.50	65.31
323	Inst. Int. de Statistique,[39] p. 447	England and Wales, 1891-1900	....	63.44	65.39
324	Inst. Int. de Statistique,[39] p. 446	Germany, 1891-1900	....	63.37	65.62
325	Inst. Int. de Statistique,[39] p. 446	Bavaria, 1901-1910	....	63.26	65.48
326	Inst. Int. de Statistique,[39] p. 446	Denmark, 1860-1869	....	63.20	66.10
327	Inst. Int. de Statistique,[39] p. 447	Italy, 1899-1902	....	63.15	66.00
328	Inst. Int. de Statistique,[39] p. 446	Wurtemberg, 1891-1900	....	62.83	65.01
329	Inst. Int. de Statistique,[39] p. 448	Sweden, 1856-1860	....	62.60	65.06
330	Inst. Int. de Statistique,[39] p. 448	Sweden, 1841-1845	....	62.60	64.95
331	Dublin and Whitney,[48] p. 442 ..	Industrial policy holders, 1911-1916. Whites. Tuberculosis present	61.7	62.5	64.7
332	Inst. Int. de Statistique,[39] p. 447	England and Wales, 1881-1890	....	62.42	64.76
333	Inst. Int. de Statistique,[39] p. 446	Prussia, 1890-1891	....	62.36	64.67
334	Inst. Int. de Statistique,[39] p. 447	Scotland, 1891-1900	....	62.32	64.90
335	Inst. Int. de Statistique,[39] p. 447	France, 1877-1881	....	62.25	65.50
336	Inst. Int. de Statistique,[39] p. 448	Sweden, 1846-1850	....	62.21	64.48
337	Inst. Int. de Statistique,[39] p. 448	Switzerland, 1889-1900	....	62.20	64.70
338	Inst. Int. de Statistique,[39] p. 446	Bavaria, 1891-1900	....	62.12	64.48
339	Bull. Met. Life Ins. Co.,[43] p. 1	Industrial policy holders, 1911-1912. Whites	61.19	62.02	64.47
340	Glover,[7] p. 100	U. S. A. Original Registration States, 1901. Foreign-born whites	61.11	61.95	64.24
341	Inst. Int. de Statistique,[39] p. 447	Scotland, 1881-1890	....	61.93	64.95
342	Inst. Int. de Statistique,[39] p. 446	Austria, 1906-1910	....	61.93	64.80

TABLE 18—*Continued*

E. General and Special Populations (Continued)

II. Females (Continued)

ITEM NO.	SOURCE	NATURE OF MATERIAL	$\mathring{e}_{15}+15$ (*years*)	$\mathring{e}_{20}+20$ (*years*)	$\mathring{e}_{30}+30$ (*years*)
343	Glover,[7] p. 108	U. S. A. Original Registration States, 1901. Whites in cities	60.93	61.86	64.45
344	Inst. Int. de Statistique,[39] p. 447	England and Wales, 1871-1880	....	61.66	64.41
345	Inst. Int. de Statistique,[39] p. 446	Germany, 1881-1890	....	61.62	64.21
346	Inst. Int. de Statistique,[39] p. 447	France, 1861-1865	....	61.60	65.10
347	Inst. Int. de Statistique,[39] p. 446	Denmark, 1835-1844	....	61.60	64.20
348	Inst. Int. de Statistique,[39] p. 448	Sweden, 1851-1855	....	61.57	63.95
349	Jones,[42] p. 29	Glasgow, 1909-1912. Two room house	60.67	61.53	....
350	Inst. Int. de Statistique,[39] p. 448	Russian Soc. Fed. Sov. Rep., 1896-1897	....	61.38	64.33
351	Inst. Int. de Statistique,[39] p. 446	Austria, 1901-1905	....	61.21	64.22
352	Inst. Int. de Statistique,[39] p. 447	Holland, 1870-1879	....	61.20	64.30
353	Inst. Int. de Statistique,[39] p. 448	U. R. S. S. European Russia, 1896-1897	....	61.16	64.14
354	Glover,[7] p. 218	Japan, 1898-1903	59.36	61.06	64.84
355	Inst. Int. de Statistique,[39] p. 447	Scotland, 1861-1870	....	61.05	64.26
356	Inst. Int. de Statistique,[39] p. 448	Switzerland, 1881-1888	....	60.97	63.76
357	Inst. Int. de Statistique,[39] p. 447	Scotland, 1871-1880	....	60.94	64.25
358	Inst. Int. de Statistique,[39] p. 446	Austria, 1895-1900	....	60.86	63.84
359	Inst. Int. de Statistique,[39] p. 446	Wurtemberg, 1876-1880	....	60.80	63.20
360	Inst. Int. de Statistique,[39] p. 448	Sweden, 1816-1840	....	60.75	63.40
361	Inst. Int. de Statistique,[39] p. 448	White Russia, 1896- 7	....	0.48	62.94
362	Inst. Int. de Statistique,[39] p. 448	Japan, 1921-1925	....	60.38	64.69
363	Inst. Int. de Statistique,[39] p. 448	Switzerland, 1876-1880	....	60.30	63.20
364	Inst. Int. de Statistique,[39] p. 447	England and Wales, 1838-1854	....	60.29	63.81

363	Inst. Int. de Statistique,[39] p. 448	Switzerland, 1876-1880	....	60.30	63.20
364	Inst. Int. de Statistique,[39] p. 447	England and Wales, 1838-1854	....	60.29	63.81
365	Inst. Int. de Statistique,[39] p. 446	Germany, 1871/72-1880/81	....	60.19	63.07
366	Inst. Int. de Statistique,[39] p. 448	Ukrainia, 1885-1898	....	60.19	....
367	Inst. Int. de Statistique,[39] p. 447	Italy, 1876-1887	....	60.00	63.40
368	Inst. Int. de Statistique,[39] p. 446	Prussia, 1867-1877	....	59.71	62.58
369	Yuan,[47] p. 167	Genealogy of a southern Chinese family. All entries	....	59.7	62.7
370	Dublin and Whitney,[48] p. 442	Industrial policy holders, 1911-1916. Colored. Tuberculosis absent	58.3	59.4	61.7
371	Bull. Met. Life Ins. Co.,[43] p. 1	Industrial policy holders, 1921. Colored	57.20	59.03	62.99
372	Jones,[42] p. 29	Glasgow, 1909-1912. One room house	57.85	58.98	....
373	Inst. Int. de Statistique,[39] p. 447	Holland, 1850-1859	....	58.92	62.16
374	Bull. Met. Life Ins. Co.,[43] p. 1	Industrial policy holders, 1930. Colored	57.26	58.70	62.01
375	Inst. Int. de Statistique,[39] p. 446	Austria, 1870-1880	....	58.28	61.52
376	Yuan,[47] p. 168	Genealogy of a southern Chinese family. Births between 1700 and 1749	....	58.0	61.5
377	Yuan,[47] p. 169	Genealogy of a southern Chinese family. Births between 1750 and 1799	....	57.6	61.1
378	Dublin,[49] p. 96	Irish born whites, living in New York State, 1910	....	57.40	....
379	Yuan,[47] p. 168	Genealogy of a southern Chinese family. Births between 1600 and 1699	....	56.9	61.4
380	Glover,[7] p. 82	U. S. A. Original Registration States, 1901. Negroes	54.79	56.89	60.70
381	Yuan,[47] p. 169	Genealogy of a southern Chinese family. Births between 1800 and 1849	....	56.8	61.1
382	Inst. Int. de Statistique,[39] p. 447	Holland, 1840-1851	....	56.72	61.03
383	Hernando,[41] pp. 176 and 177	Manila, 1920. Native born Filipinos	55.37	56.68	60.19
384	Glover,[7] p. 84	U. S. A. Original Registration States, 1901-1910. Negroes	54.05	56.17	60.09

TABLE 18—*Continued*

E. General and Special Populations (Continued)

II. Females (Continued)

ITEM NO.	SOURCE	NATURE OF MATERIAL	$\dot{e}_{15}+15$ (*years*)	$\dot{e}_{20}+20$ (*years*)	$\dot{e}_{30}+30$ (*years*)
385	Glover,[7] p. 86	U. S. A. Original Registration States, 1910. Negroes	54.18	56.14	59.61
386	Dublin and Whitney,[48] p. 442	Industrial policy holders, 1911-1916. Colored. Tuberculosis present	53.6	55.7	59.8
387	Jones,[42] p. 38	Glasgow, 1870-1872	53.32	55.31	59.35
388	Bull. Met. Life Ins. Co.,[43] p. 1	Industrial policy holders, 1911-1912. Colored	52.83	55.14	59.32
389	Glover,[7] p. 218	India, 1901-1910	45.78	47.96	52.99
390	Unweighted means	Females	61.68	63.74	66.32

[37a] Yuan, I-chin. The influence of heredity upon the duration of life in man based on a Chinese genealogy from 1365 to 1914. HUMAN BIOLOGY, Vol. 4, pp. 41-68, 1932.

[38] Approximate value got by interpolation.

[39] Office Permanent de l'Institut International de Statistique. Aperçu de la démographie des divers pays du monde. 1931. *La Haye* (Van Stockum) 1932. Pp. xxvi + 469.

[40] DePorte, J. V. Life tables for the population of New York State according to nativity. *Amer. Jour. Hyg.*, Vol. 4, pp. 302-326, 1924.

[41] Hernando, E. Life tables for the native resident population of the city of Manila for the year 1920. *Phil. Jour. Sci.*, Vol. 34, pp. 161-185, 1927.

[42] Jones, W. The Expectation of Life in the City of Glasgow. *Glasgow* (Robert Anderson) 1925. Pp. vii + 46 + 2 plates.

[43] Statistical Bulletin Metropolitan Life Insurance Company. Vol. 12, No. 12, December, 1931.

[44] *Ibid.*, Vol. 9, No. 8, 1928.

[45] *Ibid.*, Vol. 10, No. 6, 1929.

[46] Dublin, L. I., E. W. Kopf and A. J. Lotka. The components of death curves. *Amer. Jour. Hyg.*, Vol. 7, pp. 299-333, 1927.

[47] Yuan, I-chin. Life tables for a southern Chinese family from 1365 to 1849. HUMAN BIOLOGY, Vol. 3, pp. 157-179, 1931.

[48] Dublin, L. I., and Jessamine Whitney. On the costs of tuberculosis. *Quart. Publ. Amer. Stat. Assoc.*, December, 1920, pp. 441-450.

[49] Dublin, L. I. The mortality of foreign race stocks. *Sci. Monthly*, Jan. 1922, pp. 93-103.

corresponding mean figures for males in general (Item 217). Similarly the average CML (20) and the average CML (30) for *mothers* (Item 22) are *greater* than the corresponding means for women in general (Item 390), by 3.52 and 3.01 years respectively. These relations suggest, though they do not prove, that parents may enjoy some excess average longevity over and above that consequent upon their belonging to a selected class experiencing no mortality prior to ages 20 or 30. If this should prove to be the fact on the basis of more extensive experience than we are able at present to assemble, a reasonable biological explanation of it would appear possible, on the ground that parents as a class are presumably sturdier and constitutionally sounder individuals on the average than persons-not-parents, at the same ages during early adult life. There would seem to be, in short, a health factor of some importance involved in the avoidance of parenthood, and of marriage.

The mean CML (20) for fathers (Item 11) is 1.82 years *less* than the mean CML (20) for mothers (Item 22). The corresponding difference, in the same sense, between the mean CML (20)s for males and females (Items 217 and 390) is 1.99 years. The same comparison of the mean CML (30)s leads to differences of 2.62 years for parents and 1.93 years for persons in general, both differences being again in the same sense. These results indicate, so far as they go, the general actuarial normality of the material upon which sections A and B of Table 18 are based. We do not find here any such discrepancy with well established biostatistical rules as was noted earlier in discussing the biometric data.

The mean CML (20) and CML (30) for grandfathers (Item 26) are greater by 3.46 and 3.86 years respectively than the corresponding means for fathers. Similarly the grandmothers exhibit greater mean values of CML (20) and CML (30) than do the mothers, by amounts of 5.07 and 4.44 years. In short grandparents appear on the face of the case to be definitely longer-lived than parents, and correspondingly even more longer-lived than groups of persons in general living to ages 20 or 30 before experiencing any mortality. The interpretation of this finding is, however, not entirely clear. Why it is not clear will be apparent first from a logical examination of the statistical and biological elements involved in the status of parenthood and grandparenthood, and then from a realization of the dearth, or really almost complete absence, of necessary statistical information about these logically differentiated

Dublin, L. I., and Jessamine Whitney. On the costs of tuberculosis. *Quart. Publ. Amer. Stat. Assoc.*, December, 1920, pp. 441-450.

Dublin, L. I. The mortality of foreign race stocks. *Sci. Monthly*, Jan. 1922, pp. 93-103.

classes. Let us examine the case from this point of view, listing a series of propositions which appear to be true individually, inclusive as a whole, and individually mutually exclusive. Consider first the kinds of parents who are *parents-not-grandparents* at the time of record. They include:

1. Living parents all of whose children have no offspring at the time of record because they (the children) have not reached the age of puberty.

2. Living parents all of whose children have had no offspring at the time of record because they (the children) have neither married nor engaged in fruitful sexual intercourse.

3. Living parents all of whose children have had no offspring at the time of record because they (the children) are effectively sterile (as evidenced by the fact of completed childless marriage—♀ past the menopause and/or ♂ more than 60 years of age, since the number of children engendered by men over 60 is negligibly small).[81]

4. Living parents all of whose children have had no offspring at the time of record because they (the children) were divorced before producing any, and have not remarried.

5. Living parents all of whose children have had no offspring at the time of record because they (the children) were widowed before producing any, and have not remarried.

6. Living parents all of whose children have had no offspring at the time of record because they (the children) have previously died childless.

7. Living parents whose children are, as a sibship, some combination of 1-6 inclusive.

8. Dead parents all of whose children have had no offspring at the time of record because they (the children) have not reached the age of puberty.

9. Dead parents all of whose children have had no offspring at the time of record because they (the children) have neither married nor engaged in fruitful sexual intercourse.

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[81] In the U. S. Registration Area the number of births in any given year sired by all fathers 55 years or over in age is usually less than three-quarters of one per cent of the total births. Unfortunately the Census Bureau does not separately tabulate ages of fathers above 55, but by the time 60 is reached the proportion of children sired must be extremely small, and for all practical statistical purposes entirely negligible.

10. Dead parents all of whose children have had no offspring at the time of record because they (the children) are sterile (as evidenced by the fact of completed childless marriage— ♀ past the menopause and/or ♂ more than 60 years of age).

11. Dead parents all of whose children have had no offspring at the time of record because they (the children) were divorced before producing any, and have not remarried.

12. Dead parents all of whose children have had no offspring at the time of record because they (the children) were widowed before producing any, and have not remarried.

13. Dead parents whose children are, as a sibship, some combination of 8-12 inclusive.

We have next to consider the classes of *parents-who-are-not-and never-can-become-grandparents.* They include:

14. Living parents who never can be grandparents because all of their children never married nor engaged in fruitful sexual intercourse and were, at the time of record, past the physiological age of fertility as defined in 3 *supra.*[51a]

15. Living parents who never can be grandparents because they are themselves past the physiological age of fertility as defined in 3 *supra,* and all their children were divorced before producing offspring, and did not remarry, if at all, until after they (the children) had passed the physiological age of fertility as defined in 3 *supra.*

16. Living parents who can never be grandparents because they are themselves past the physiological age of fertility, and all their children were widowed before producing offspring, and did not remarry, if at all, until after they (the children) had passed the physiological age of fertility as defined in 3 *supra.*

17. Living parents who never can be grandparents because they are themselves past the physiological age of fertility as defined in 3 *supra,* and their children have all died childless.[51a]

18. Living parents who never can be grandparents because, as a group, they and their children present together some combination of the specifications included in 14-17 inclusive, and no other.

[51a] It should be noted that logically Class 14 is a sub-group of Class 2, but for practical statistical reasons it seems advisable to list them as separate classes. The same thing is true of Classes 17 and 6, and Classes 19 and 9.

19. Dead parents who never can become grandparents because all of their children never married nor engaged in fruitful sexual intercourse and were, at the time of record, past the physiological age of fertility as defined in 3 *supra*.[51a]

20. Dead parents who never can become grandparents because all their children were divorced before producing offspring, and did not remarry, if at all, until after they (the children) had passed the physiological age of fertility as defined in 3 *supra*.

21. Dead parents who never can become grandparents because all their children were widowed before producing offspring, and did not remarry, if at all, until after they (the children) had passed the physiological age of fertility as defined in 3 *supra*.

22. Dead parents who never can become grandparents because all their children died childless.

23. Dead parents who never can become grandparents because, as a group, they and their children present together some combination of the specifications included in 19-22 inclusive, and no other.

It is believed that categories 1-23 inclusive comprise all types of *parents-not-grandparents* capable of being statistically differentiated relative to the variables considered. Of these classes, 3, 10, and 14-23 inclusive are *parents-never-grandparents,* while the remaining classes are *merely-parents-not-grandparents-at-the-time-of-record*. The parents-never-grandparents are persons who, while they have been parents, can never achieve the status of grandparenthood under any circumstance, being debarred therefrom by some one or a combination of the stated specifications. The merely-parents-not-grandparents-at-the-time-of-record are persons who conceivable still have the *possibility* of achieving grand-parenthood at some future time.

It is clearly to be understood that the variables considered in the above array of logical categories are by no means all of those that may be involved in determining the status of persons relative to parenthood and grandparenthood. But they do include those of most importance in dealing with actual statistical data in work on the problems of longevity.

Let us now turn to the consideration of grandparents. Since a grandparent is necessarily always a parent it follows that the classificatory scheme used above needs only to be continued to include the final possible class of parents, namely *parents-also-grandparents*. Of

these it is necessary for general statistical purposes to differentiate but six broad classes, *viz.*:

24. Living parents-also-grandparents, all of whose children are living at the time of record.

25. Living parents-also-grandparents, all of whose children are dead at the time of record.

26. Living parents-also-grandparents having both living and dead children at the time of record.

27. Dead parents-also-grandparents, all of whose children are living at the time of record.

28. Dead parents-also-grandparents, all of whose children are dead at the time of record.

29. Dead parents-also-grandparents, having both living and dead children at the time of record.

We have noted above the difficulty of interpretation of the observed greater average CML (20) and CML (30) of grandparents over parents and over cohorts of persons in general in which no mortality had occurred before ages 20 and 30 respectively. The fundamental reason for this difficulty can now be precisely stated. It is that, so far as we are aware, *there does not exist,* for any population, anywhere, exact and comprehensive information as to either (a) *the frequency* in the population, or (b) the *age distribution* of any one of the 29 statistically differentiated types of parents and grandparents listed above. Until such information is available it is impossible to go much below the surface in the analysis of the genetics of longevity. It is our hope to be able in the not too distant future to publish a detailed study that will show, for a fairly large and random population sample, (a) the frequency of occurrence, absolute and relative, (b) the age distribution, and (c) the realized fertility of each of the 29 categories of parents (and grandparents) listed above.

Returning now to Table 18, let us consider briefly some points brought out in Section E, parts I (male) and II (female). Focussing attention upon the CML (20) column, it is seen that the range for males is from 47.46 years mean expected duration of life to 69.40 years. The lowest male figure is for India (1901-1910) and the highest for Denmark (1921-1925). The difference between these two extremes is 21.94 years. Back of this large difference in average longevity lie differences in many other variables, including race, climate, education, hygiene

and sanitation, food habits, density of population, etc. Most of these differences can be more or less easily rationalized as the sort of difference that would be expected to lead to the observed difference in average expectation of life at birth. But it is also worthy of note that Denmark has nearly, if not quite, the highest per capita consumption of alcohol of any country in the world, while India has one of the lowest per capita alcohol consumptions.

It is interesting further to note that the countries whose life tables give male CML (20) values of 66 years and above, are (with only one real exception—White Russia) countries populated predominantly with Nordic stocks. On the other hand, the population groups exhibiting CML (20) values below 56 years are either Negroes, Filipinos, Chinese, Irish, or East Indians, or are primarily Nordic stocks existing under the conditions of public health and sanitation prevailing from a half-century to a century ago.

For the females the range in CML (20) values is from 47.96 years —again for India 1901-1910—to 70.36 years, the computed average longevity of women in New Zealand, 1921-1922. The difference is 22.40 years. In general the racial distribution of the CML (20) values for the females follows closely that for the males, as would naturally be expected.

Figure 16 shows the percentage frequency distribution of the CML (20) values in Section E of Table 18.

The diagrams of Figure 16 show clearly the somewhat skew character of the distributions, and the general superiority of the females over the males interracially in longevity.

F. The Siblings of the Longevous

Up to this point the analysis has been concerned with TIAL and its component elements. It seems desirable in a final section before proceeding to the discussion of the correlations to present some data regarding the sibs of the propositi in the Long. Series and the FHR Series.

Owing to failure of complete recording of the sex of siblings in the original records three cases in the Long. Series and six cases in the FHR Series had to be omitted from the sibling study, leaving for the

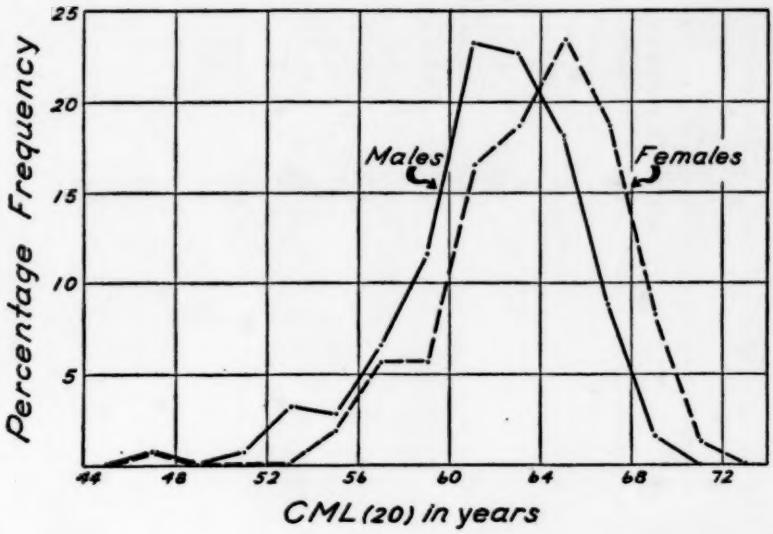

FIG. 16. FREQUENCY POLYGONS SHOWING THE PERCENTAGE DISTRIBUTION OF CML (20)—THAT IS, COMPUTED AVERAGE LONGEVITY OF POPULATIONS EXPERIENCING NO MORTALITY BEFORE AGE 20—VALUES FOR MALES (SOLID LINE) AND FEMALE (DASH LINE), FROM THE LIFE TABLE VALUES OF SECTION E OF TABLE 18

tables in this section of the investigation 362 sibships in the Long. Series and 137 in the FHR Series. Since the parents of the propositi (and therefore of their sibs) in both series are dead, it is evident that the sibships are complete, and their size represents the completed total realized fertility of the matings from which they came.

Tables 19 and 20 show, for the Long. Series and the FHR Series respectively, the total size of the sibship to which the propositus belonged (*including* the propositus in each case), and the sex-ratio (expressed as percentage of males to total number in the sibship).

It appears that in six cases (1.7 per cent) in the Long. Series, and five (3.6 per cent) in the FHR Series the propositus was an only child. From one the size of the sibship ranges to 16 in the Long. Series data, and to 24 in the FHR Series. The distributions of sibship size are shown graphically in Figure 17.

TABLE 19

Correlation Table for (a) Male Sex-ratio and (b) Total Size of Sibships of Which the Propositi in the Long. Series Were Members

TOTAL NUMBERS IN SIBSHIP	PERCENTAGE OF ♂♂											ABSOLUTE TOTALS	PERCENTAGES
	0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100		
1	3	..	..	..	..	..	..	..	..	..	3	6	1.7
2	2	..	..	..	..	6	..	..	..	..	4	12	3.3
3	2	..	..	9	..	..	14	..	..	..	4	29	8.0
4	1	..	12	..	..	14	..	10	..	..	3	40	11.0
5	1	..	8	..	12	..	14	..	4	..	3	42	11.6
6	..	2	..	10	..	7	6	..	2	..	1	28	7.7
7	..	2	5	..	13	17	..	9	2	..	..	48	13.3
8	..	1	8	5	..	14	7	5	1	..	..	41	11.3
9	..	1	2	5	9	15	10	3	1	..	..	46	12.7
10	..	..	..	..	4	5	3	1	2	..	..	15	4.1
11	2	..	1	5	2	2	5	4	2	..	..	23	6.4
12	..	..	3	7	1	4	4	1	..	1	..	21	5.8
13	..	..	..	3	..	..	..	1	1	..	..	5	1.4
14	..	..	..	..	..	3	..	..	..	..	..	3	.8
15	..	..	..	..	..	1	..	1	..	..	..	2	.6
16	..	..	..	..	..	1	..	..	..	..	..	1	.3
Totals	11	6	39	44	41	89	63	35	15	1	18	362	100.0

TABLE 20

Correlation Table for (a) Male Sex-ratio and (b) Total Size of Sibships of Which the Propositi in the FHR Series Were Members

TOTAL NUMBERS IN SIBSHIP	PERCENTAGE OF ♂ ♂											ABSOLUTE TOTALS	PERCENTAGES
	0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100		
1	3	..	..	..	..	..	..	..	..	..	2	5	3.6
2	4	..	..	..	..	5	..	..	..	..	1	10	7.3
3	4	..	..	6	..	..	4	..	..	..	1	15	10.9
4	1	..	4	..	..	6	..	3	..	..	1	15	10.9
5	..	..	2	..	6	..	9	..	4	..	..	21	15.3
6	..	..	..	3	..	5	2	..	1	..	..	11	8.0
7	..	..	3	..	2	4	..	5	1	..	..	15	10.9
8	..	..	..	1	1	3	5	1	1	..	..	12	8.8
9	..	..	1	1	1	1	1	1	..	..	..	6	4.4
10	..	..	..	..	2	4	1	3	..	..	..	10	7.3
11	..	..	..	1	..	1	2	..	..	..	..	4	2.9
12	..	..	1	1	1	1	2	1	..	..	..	7	5.1
13	..	..	..	..	..	..	..	..	..	..	..	..	...
14	..	..	..	..	..	1	1	..	..	..	..	2	1.5
15	..	..	..	..	2	..	..	1	..	..	..	3	2.2
———	—	—	—	—	—	—	—	—	—	—	—	—	———
24	..	..	..	..	..	1	..	..	..	..	..	1	.7
Totals	12	..	11	13	15	32	27	15	7	..	5	137	99.8

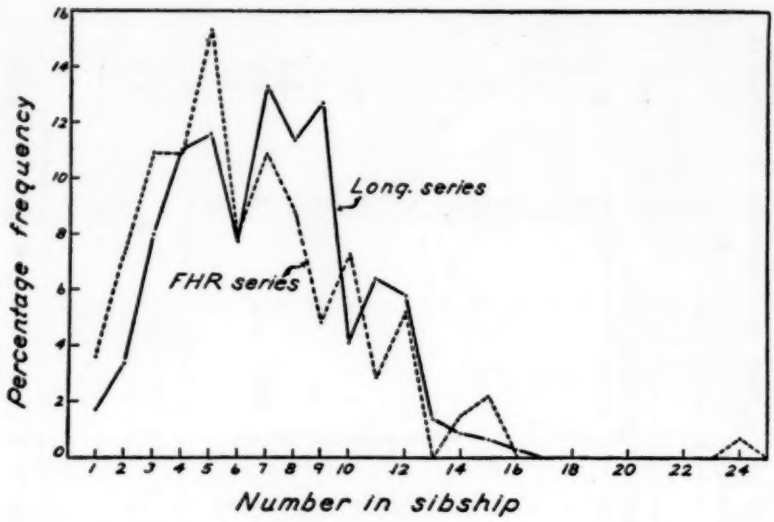

FIG. 17. FREQUENCY POLYGONS FOR SIBSHIP SIZE

The biometric constants for size of sibship and sex-ratio are shown in Table 21.

TABLE 21

Biometric Constants for Size of Sibship and Sex-ratio

	SIZE OF SIBSHIP		SEX RATIO	
	Long. Series	FHR Series	Long. Series	FHR Series
Mean	7.02± .11	6.43± .21	53.31± .76	52.55±1.31
Median	7.00± .14	5.73± .26	54.49± .95	55.47±1.64
Standard deviation..	3.03± .08	3.62± .15	21.35± .54	22.75± .93
Coefficient of variation	43.18±1.27	56.35±2.95	40.05±1.16	43.29±2.08

The mean and the median sibships are somewhat larger in the Long. Series than in the FHR Series. The difference between the means cannot be regarded as significant in comparison with its probable error; that between the medians is larger and according to the conventional usage probably is statistically significant. The distributions of Figure

17 indicate clearly that there is a definite tendency of the sibships to run larger in the Long. Series than in the FHR Series. This tendency towards larger sibship sizes in the Long. Series suggested in Table 21 is consonant with the idea that great longevity, individual or familial, is one mark of high general biological vigor and strength in the stock, which may also express itself in high fertility. The longevous person or family appears to be biologically superior in a variety of respects.

The Long. Series sibships are somewhat less variable in size, both absolutely and relatively, than the FHR Series sibships, but by amounts so small as to be of no great moment biologically, in our view.

Again the male sex ratio appears to be about normal and not markedly different in the two series of data, either in magnitude or variability. The slight excess male production and lower variability in the Long. Series probably cannot be regarded as significant.

Altogether we conclude that the longevous families and those of the FHR Series are substantially similar in respect of the two biologically important variables fertility and sex ratio, with a somewhat greater fertility in the longevous families.

It is plain from inspection of the tables that there is no marked degree of correlation between the two variables sex-ratio and size of sibship.

The present data give no strong support to the interesting and ingenious theory of Pitt-Rivers [52] that increasing masculinity is associated with population decline in numbers. An expected corollary of this view would be that in groups showing relatively higher masculine sex-ratios there would be associated relatively lower fertilities, and *vice-versa,* and generally a negative or inverse correlation between male sex-ratio and fertility. The present material is, however, not strictly pertinent to the elucidation of Pitt-Rivers' point, because the fertilities and the sex-ratios of Tables 19 and 20 appertain to the same sibships, while presumably what is needed is the sex-ratios of the *parental* sibships (which are not here tabled) and the fertilities of a group of parents from those sibships (which are here tabled). At the same time it might not be wholly unreasonable to expect a negative correlation in

[52] Pitt-Rivers, G. H. L.-F. The Clash of Culture and the Decline of Races. *London* (Routledge) 1927. Pp. xiv + 312. See especially the "Supplement on Sex-ratio," pp. 243-275.

the present material, if the theory were operating, because of the known fact that fertility is in some degree inherited.

We may turn our attention next to the *mortality* that has been experienced in the sibships in the Long. Series and the FHR Series. A complete actuarial analysis of these sibships will not be undertaken here, for two reasons. The first is that it is not essential for the purposes of the present study; the second is that we intend later to construct complete life tables for the sibships to which nonagenarians and centenarians belong, using the whole of our material, which is much more extensive than the present Long. Series sample limited by the TIAL restrictions, as has already been pointed out. It will suffice for present purposes to show the distributions of the living and the dead in the

TABLE 22

Absolute and Percentage Distributions by Age (For All Persons of Recorded Age) of the Dead and Living at the Time of Record in the Sibships to Which the Propositi Belong in the Long. Series and the FHR Series

AGE (*years*)	LONG. SERIES				FHR SERIES			
	No. dead under stated age	Per cent dead	No. still living under stated age	Per cent still living	No. dead under stated age	Per cent dead	No. still living under stated age	Per cent still living
10	252	11.5	0	0	188	20.8	2	.22
20	322	14.8	0	0	226	24.9	16	1.8
30	444	20.3	0	0	285	31.5	57	6.3
40	553	25.3	0	0	329	36.3	179	19.8
50	644	29.5	1	.05	362	40.0	320	35.3
60	744	34.1	4	.18	382	42.2	440	48.6
70	903	41.4	15	.69	389	42.9	498	55.0
80	1225	56.1	55	2.5	389	42.9	516	57.0
90	1571	72.0	131	6.0	389	42.9	517	57.1
100	1666	76.3	458	21.0	389	42.9	517	57.1
110	1667	76.4	516	23.6	389	42.9	517	57.1

Long. and the FHR Series, as is done in Table 22, and then in Table 23 present the biometric constants for age in the various groups of siblings.

From Table 22 the following points are to be noted:

1. In the Long. Series the mortality is absolutely completed at all ages below 40 years, since there remain no persons still living below those ages, and is practically completed at all ages below 80 years, since only 55 persons (or only 2.5 per cent of the total) are still living at ages below 80. The case is quite different in the FHR Series where at no age (considering only 10 year classes) can the mortality be regarded as theoretically complete, since there are still some living persons in each age class up to 90 years.

2. But even though the FHR Series mortality is incomplete at the lower ages, while the Long. Series mortality is complete at the same ages, *the former is much higher than the latter,* at the time of record. Thus up to age 10, in the FHR Series 20.8 per cent of the sibs died, while in the same age period only 11.5 per cent of the Long. Series sibs died.

3. Consider the situation up to age 40. In this period 25.3 per cent of the total number of children ever born in the Long. Series sibships died, leaving 74.7 per cent still surviving at age 40. This compares with a life table percentage of 65.9 surviving at age 40.[7] In this same life table (Both Sexes, Orig. Reg. States, 1901) the age at which 74.7 per cent of all individuals are still surviving is approximately 25 years, instead of the 40 years of the Long. Series sibships. In other words, *the individuals in sibships to which our nonagenarians and centenarians belonged experienced a much lower mortality below age 40 than do people in general.* Up to 40 years of age the FHR Series sibships had experienced an *incomplete* mortality of 36.3 per cent, and still had 19.8 per cent of all those born living at ages below 40 available for further dying. It seems clear beyond question or doubt that, insofar as mortality is an index, the propositi of the Long. Series belong to stouter stocks, biologically considered, than do those of the FHR Series.

The *infant mortality* record (deaths under one year of age) in the Long. Series sibships is so low as to be worthy of special discussion. In these sibships there were 2183 persons born. Of these there died in the first year of life (i.e., between birth and one year of age) 128, or *5.86 per cent.* In the life table referred to above the deaths in the first year of life amounted to 12.45 per cent. In other words the sibships to

TABLE 23

Biometric Constants for Age of the Various Groups of Siblings

GROUP	MEAN (*years*)	MEDIAN (*years*)	STANDARD DEVIATION (*years*)	COEFFICIENT OF VARIATION (*per cent*)
a. Living sibs of propositi. Long. Series.....	81.36± .51	82.70± .64	9.17± .36	11.27± .44
b. Living sibs of propositi. FHR Series.....	44.71± .46	44.27± .57	13.29± .33	29.72± .79
c. All living sibs, including propositi. Long. Series	92.13± .27	93.88± .34	9.06± .19	9.83± .21
d. All living sibs, including propositi. FHR Series	45.77± .41	45.64± .51	13.70± .29	29.93± .68
e. Dead sibs of propositi. Long. Series.....	54.33± .51	65.63± .64	30.65± .36	56.41± .84
f. Dead sibs of propositi. FHR Series.....	17.89± .64	11.71± .80	18.65± .45	104.2± 4.5

which our nonagenarians and centenarians belong experienced an infant mortality ***less than half that experienced by the general population roughly some 70 years later*** when infant mortality rates generally had been considerably reduced as result of greater knowledge as to infant care, and generally better hygiene and sanitation. As a matter of fact the total mortality in the Long. Series sibships up to age 10 was less than that of the general population during the first year of life in 1901. In the FHR Series sibships the mortality in the first year of life was 14.35 per cent, a figure not widely different from that of the general population in 1901. Nothing could show more clearly the importance of sound heredity in respect of longevity as a factor in the reduction of infant mortality than the experience of these Long. Series sibships.

Table 23 gives the biometric constants for age of the various groups of siblings.

Table 23 confirms in another way the conclusions already reached from the figures of Table 22. The Long. Series sibships exhibit an enormously greater store of vitality, as expressed in duration of living up to the time of record, than do the FHR sibships. This remains true after all allowance has been made for the fact that in the nature of the case the FHR sibships are necessarily farther away from the completion of their total living than are those of the Long. Series.

It is of interest to note that while the difference in mean ages (living) of the propositi in the two series is 46.68 years (*vide* Table 3 *supra*) the difference in mean age between the living siblings of the propositi in the two series is only 36.65 years. In the Long. Series the mean age of the living siblings of the propositi is 85.3 per cent of the mean age (living) of the propositi themselves. In the FHR Series the corresponding percentage is 91.7. The extremely longevous person tends to be exceptional, even in his own sibship. The mean age of the dead siblings in the Long. Series sibships is 59.0 per cent of the mean age of the living siblings (including the propositi) in the same series, while the corresponding percentage for the FHR Series is 39.1. This is another expression of heavy mortality at early ages in the FHR Series as compared with the Long. Series, which has been noted above.

The mean individual total longevity, realized by the dead and expected according to the life table by those still living at the time of record, is for the 2183 members of the Long. Series sibships, 63.88 years, or 14.64 years greater than the expected mean duration of life at birth from the life table for the Original Registration States in 1901

(both sexes). The corresponding figure for the 906 members of the FHR Series sibships is 47.62 years, or 1.62 years less than the life table expectation of life at birth, an insignificant difference.

From Table 10 we have seen that the difference in mean TIAL between the two series is 60.69 years. Hence it may be deduced that, in the two samples here dealt with, an excess in mean TIAL per pedigree of 60.69 years led to an average excess of longevity in the offspring of 16.26 years, or that, on the average, each additional 3.7 years in mean TIAL of the Long. Series over the FHR Series had associated with it one additional year in mean duration of life of the offspring.

G. Correlations

The correlations in respect of age at death between the various sorts of individuals, or combinations of them, who contribute to TIAL, will necessarily fall into two broad categories, viz.:

a. Homogamic correlations,

measuring the degree of assortative mating between the two sides of the pedigree at various levels. These correlations will throw light upon the question as to how marked the tendencies were in the two series (Long. and FHR) for the immediate ancestors of the propositi to take mates like themselves in respect of duration of life. That is, did persons who in the end achieved relatively great longevity choose in early life mates who also turned out to be relatively highly longevous? And similarly for relatively short-lived persons? It is just such assortative choosing of mates which must take place if high homogamic correlations in respect of longevity are to arise.

b. Kinship (genetic) correlations,

measuring the degree of resemblance between kin in lineal descent, as between father and son for example. It is a cardinal tenet of faith of the Galton-Pearson school of genetic thought that such correlations measure the intensity of inheritance, and do so entirely objectively—uninfluenced logically by any theoretical considerations as to whether mechanism of the hereditary process involved is Mendelian or something else.

With the material available in the present study it is possible to set up under both categories *a* (homogamic) and *b* (kinship) certain correlations that, in the nature of the case, are either:

a. Sensible; that is, make sense biologically.

β. Not sensible; that is, are nonsense correlations biologically.

We propose to exhibit and compare both kinds.

a. Homogamic Correlation

Table 24 shows the sensible assortative mating correlations that can be set up from the available material, dealing with single individuals. In this and all following tables of correlation coefficients the subscripts to r the correlation coefficient refer to the position of the individuals (or combinations of individuals) involved, as set forth in the pedigree shown in Figure 1 *supra*.

TABLE 24

Theoretically Sensible Simple Homogamic Correlations in Respect of Longevity

CORRELATION BETWEEN AGE AT DEATH OF	LONG. SERIES	FHR SERIES
1. Father [53] and mother.[53] r_{ef}	+ 0.068 ± .035	+ 0.403 ± .047
2. Father's father [53] and father's mother. r_{ab} ..	+ .239 ± .033	+ .126 ± .056
3. Mother's father and mother's mother. r_{cd} ..	+ .069 ± .035	+ .100 ± .056
Means	+ .125	+ .210

We know of no biological reason why all six coefficients of correlation in Table 24 should not be substantially equal to each other. That is to say, if homogamy relative to longevity is a real factor in the selection (conscious or unconscious) of mates for marrying amongst human beings, as has been asserted, then there would seem to be no reason to suppose that its intensity would not be reasonably constant in fairly

[53] Throughout the discussion of correlation, that is in Tables 24-30 inclusive, the designations of persons correlated are *by their kinship to the propositus*. That is to say, in these tables "father" means "father of the propositus," and so similarly for all other cases *mutatis mutandis*. Furthermore if the reader will refer back to Figure 1, and pay attention to the subscrips of r in the correlation discussion, it will be clear at all times what individuals are being correlated.

homogeneous groups of people over so short a time span as a single generation. But actually there is a considerable diversity amongst these six coefficients. Four of the six are not significantly differen zero. The other two are quite large, as homogamic correlations go. The mean value for the whole six is 0.168, and the standard deviation 0.120, leading to a coefficient of variation of 71.4 per cent indicative of a rather highly variable state of affairs. Attention should be called to the fact that the high coefficient of +0.403 for the parents in the FHR Series may owe a part of its magnitude to the manner in which the FHR Series was selected, as described in an earlier section of this study. The rule of formation of the group included *inter alia* the requirement that both parents should be dead, and the propositus living. This would have some tendency to make the two parents alike in age at death.

But before discussing further the correlations of Table 24 let us examine the nonsense correlations of Table 25.

TABLE 25

Theoretically Nonsense Simple Homogamic Correlations in Respect of Longevity

CORRELATION BETWEEN AGE AT DEATH OF	LONG. SERIES	FHR SERIES
Father's father with mother's mother. r_{ad}	+ 0.078 ± .035	+ 0.126 ± .056
Mother's father with father's mother. r_{cb}	+ .127 ± .035	+ .039 ± .056
Means	+ .103	+ .083

Taking a broad view of the matter it seems open to some question as to whether the coefficients of Table 25 are widely enough different in their values from those of Table 24 as to indicate any real and significant assortative mating in respect of longevity in this material. The fluctuation of random sampling would seem to have been a considerable factor in the production of the values in the two tables. Of course there may be some prepared to allege that the correlations of Table 25 are not nonsense correlations; that persons do in fact choose their mates upon the basis that his father's longevity, and his intended wife's

mother's longevity are similar, and *vice versa*. Perhaps such an allegation is not nonsense, but it seems so to us.

Let us examine next what may be called a compound homogamic correlation, and another compound correlation that may be compared with it. These values are shown in Table 26.

TABLE 26

Compound Homogamic Correlation in Respect of Longevity

VARIABLES CORRELATED	LONG. SERIES	FHR SERIES
(Summed longevity of all three immediate male ancestors) and (Summed longevity of all three immediate female ancestors)		
$r_{(a+c+e)\ (d+b+f)}$	+0.190±.034	+0.315±.051
(Summed longevity of all three immediate ancestors of propositus on father's side) and (Summed longevity of all three immediate ancestors of propositus on mother's side)		
$r_{(a+b+e)\ (c+d+f)}$	+0.107±.035	+0.143±.055

While the correlations of Table 26 are by no means very high, still this table makes a different impression than does either Table 24 or Table 25. The coefficients range from 2.6 (in the worst case) to 6.2 (in the best case) times the probable errors. The correlations of the first line of the table (all male with all female ancestors) are respectively 5.6 and 6.2 times their probable errors. So that it would seem that in both the Long. and the FHR Series there is a statistically significant correlation between the three immediate male ancestors of the propositus and the three immediate female ancestors, in regard to their respective summed longevities. This correlation appears to be biologically sensible. It is a little difficult to say whether the correlations of the second line of Table 26 are to be regarded as making sense or nonsense. It depends on the viewpoint. These two correlations, $r_{(a+b+e)\ (c+d+f)}$, measure the degree of likeness of the two sides of the pedigree in respect of summed longevity. This surely is an interesting

and sensible thing to measure, considered in and of itself. But on the other hand, it is not wholly or strictly an assortative mating or homogamic correlation, and insofar as it is not it is a nonsense correlation relative to homogamy. The actual values of the coefficients are on the borderline of statistical significance (3.1 and 2.6 times their probable errors) and cannot be asserted to be certainly outside the range of chance fluctuation.

A point that ought not to be left without comment is that in Tables 24 and 26 there is an evident tendency for the coefficients to be larger in the FHR Series than in the Long. Series. In the 5 pairs of comparable coefficients the one for the FHR Series is the larger in all but one case. The differences are, to be sure, mostly insignificant statistically in their magnitudes. But the trend seems to be remarkable in that it is opposite to what might be expected *a priori*. If ancestral assortative mating in respect of longevity plays any part in the production of great duration of life, either absolute or relative, in the propositus, then it might reasonably be expected that the homogamic correlations would tend to be higher in the Long. Series than in the FHR Series. But the trend of the actual figures is rather the opposite. As has already been pointed out some part of this result may be due to the manner of formation of the FHR Series. But this consideration would not be likely to affect sensibly the grandparental homogamic correlations.

Many years ago Romanes stressed the fact that homogamy is, in the very nature of the case, an important factor in organic evolution. In fact it is difficult to see why its significance is not potentially as great as that of natural selection, and so far as concerns civilized man perhaps even greater. But it is only in comparatively recent years that any attempt has been made seriously and systematically to measure the extent and degree of assortative mating.

We shall not attempt to review the literature on homogamy in man in any systematic way here, partly because of lack of space and partly because much of it is irrelevant to the present purpose. It may, however, be noted in passing, as evidence we hope of increasing interest in the subject, that Jones,[84] who has reviewed the subject, finds that when the most reliable intelligence tests are used the homogamic correlation for intellectual abilities tends to take values of about +0.5. Willough-

[84] Jones, H. E. Homogamy in intellectual abilities. *Amer. Jour. Soc.*, Vol. 35, pp. 369-382, 1929.

by,[55] an active worker in this field, has studied five modern population groups and two historical populations relative to the degree of homogamy in respect of fertility. He finds that in his modern population groups the fertility homogamic correlations range from —0.13 to values of the order of +0.30, with a tendency for higher degrees of homogamy in the economically superior groups. Crux and Haeger[56] reach the unexpected, and so far as we are aware unconfirmed conclusion that normally mate choice in man follows the rule of opposites in respect of bodily habitus, the pyknic tending to choose the asthenic, and *vice versa.* They allege that deviations from this rule usually indicate personalities injured by unfortunate childhood experiences. It is difficult to reconcile this conclusion with the results of Pearson and Lee,[57] Rosinski,[58] and Willoughby[58a] who find significant positive correlations between husband and wife in respect of a number of physical characters of the body, each taken singly. Thus we have the following values:

Character	*Pearson and Lee*	*Rosinski*	*Willoughby*
Stature	+0.280±.019	+0.138	+0.16
Span	+ .199±.020	.....	.....
Forearm length	+ .198±.021	.....	.....
Face width	.....	.....	+ .15
Form of lips	.....	+ .475	.....
Head width	.....	.....	+ .10
Head length	.....	.....	+ .06
Form of nose	.....	— .177	.....
Facial index	.....	+ .076	.....
Face width index	.....	.....	+ .08
Nasal index	.....	— .066	.....
Cephalic index	.....	+ .006	+ .08

[55] Willoughby, R. R. Homogamy in fertility. *Eug. Rev.,* Vol. 23, pp. 223-229, 1931.

[56] Crux, J. and F. Haeger. Körperbau und Gattenwahl. *Ztschr. f. Sex.-wiss. u. Sex.-pol.,* Bd. 17, pp. 337-348, 1930.

[57] Pearson, K. and Alice Lee. On the laws of inheritance in man. I. Inheritance of physical characters. *Biometrika,* Vol. 2, pp. 357-462, 1903.

[58] Rosinski, B. Anthropogenetische Auslese. *Anthrop. Anz.,* pp. 49-64, 1929.

[58a] Willoughby, R. R. Somatic homogamy in man. HUMAN BIOLOGY, Vol. 5, pp. 690-705, 1933.

The pioneer investigation regarding homogamy in respect of duration of life, and still the most extensive one, is the study made over 30 years ago by Pearson [59] and his co-workers on English tombstone and genealogical records. The homogamic correlations obtained were remarkably consistent, and ran as follows:

Correlation Between Husband and Wife in Age at Death (Pearson)

Wensleydale and District (Tombstones)	+0.2200 ± .0244
Oxfordshire (Tombstones)	+ .2500 ± .0211
London (Tombstones)	+ .4204 ± .0176
Society of Friends (Genealogies)	+ .1999 ± .0212

The London value is thought to be too high and is excluded in forming the average figure of +0.2233. Our mean value of +0.210 for the FHR Series (Table 24) is in rather close agreement with this. But even so we are still somewhat reluctant, in view of the very considerable variation shown by our coefficients and their disturbing nearness to comparable nonsense correlations, to accept as demonstrated the reality of a sensible and significant assortative mating in man in respect of longevity. It would seem prudent to wait for more extensive and confirmatory evidence before accepting such a conclusion unreservedly. Stoessiger's [26] recent study of Pearson's family data gives a lower value (+0.1564 ± .0197) than those cited above from the earlier work.

Kinship (Genetic) Correlations

In Table 27 are presented all of the possible theoretically sensible simple kinship correlations that can be derived from the present material.

In but one of the eight parent-offspring correlations exhibited in Table 27 is the coefficient three or more times its probable error. But before discussing further the data of this table we wish to present the corresponding nonsense correlations—nonsensical because it cannot be reasonably alleged that a man or woman inherits anything biologically from his or her *consort's* parents.

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[59] Assortative mating in man. A coöperative study. *Biometrika*, Vol. 2, pp. 481-498, 1903.

TABLE 27

Theoretically Sensible Parent-Offspring Correlations in Respect of Longevity

CORRELATION IN RESPECT OF AGE AT DEATH BETWEEN	LONG. SERIES	NO. OF PAIRS
Father's father and father (Father and son type) r_{ae}	+0.093±.035	365
Father's mother and father (Mother and son type) r_{be}	+ .157±.034	365
Mother's father and mother (Father and daughter type) r_{cf}	+ .016±.035	365
Mother's mother and mother (Mother and daughter type) r_{df}	+ .067±.035	365
Mean numerical values (regardless of signs)	.083	
	FHR SERIES	**NO. OF PAIRS**
Father's father and father (Father and son type) r_{ae}	+0.086±.056	143
Father's mother and father (Mother and son type) r_{be}	— .006±.056	143
Mother's father and mother (Father and daughter type) r_{cf}	+ .159±.055	143
Mother's mother and mother (Mother and daughter type) r_{df}	+ .003±.056	143
Mean numerical values (regardless of signs)	.064	

Having regard to the probable errors involved there is plainly little to choose between sensible Table 27 and nonsensical Table 28, so far as the intensity of correlation, as measured by the coefficients, is concerned. In point of fact the four nonsense coefficients have actually a higher average numerical value than the four sensible coefficients, in the case of the FHR Series.

Furthermore if Table 27 be compared with Table 24 it is seen that the mean sensible homogamic correlations are actually higher (though

TABLE 28

Theoretically Nonsense Parent-offspring Correlations in Respect of Longevity

CORRELATION IN RESPECT OF AGE AT DEATH BETWEEN	LONG. SERIES	NO. OF PAIRS
Father's father and subject's mother, r_{af}	—0.022±.035	365
Father's mother and subject's mother, r_{bf}	— .061±.035	365
Mother's father and subject's father, r_{ce}	+ .033±.035	365
Mother's mother and subject's father, r_{de}	+ .032±.035	365
Mean numerical values (regardless of signs)	.037	
	FHR SERIES	NO. OF PAIRS
Father's father and subject's mother, r_{af}	+0.045±.056	143
Father's mother and subject's mother, r_{bf}	— .147±.055	143
Mother's father and subject's father, r_{ce}	+ .050±.056	143
Mother's mother and subject's father, r_{de}	+ .070±.056	143
Mean numerical values (regardless of signs)	.078	

not significantly so) than the mean sensible parent-offspring correlations in both series. This surely is an odd state of affairs, in view of the fact, which we believe has been abundantly demonstrated by the evidence presented in this study, that heredity plays an important part in the biological determination of longevity.

Tables 29 and 30 present respectively sensible and nonsense compound kinship (parent-offspring) correlations, which are in fact equivalent to correlations between an individual and his mid-parent, in the Galtonian sense of the word.[60]

[60] Galton took as mid-parental values for correlational purposes the mean of the two parental values of the variable concerned, making where necessary a sex-correction to bring female measures into equivalence with male. It can make no essential difference in the results so far as concerns *correlation* if one takes (father + mother)/2, or simply (father + mother) as we have done here.

It is apparent that the compound or mid-parent-offspring correlations are not substantially different from the simple parent-offspring correlations of Table 27. There is the same considerable variation, and the same lack of convincing evidence of any general or real departure from zero correlation.

The nonsense correlations of Table 30 are somewhat smaller on the whole, than the sensible correlations of Table 29, but not by amounts likely to convince anyone that the latter are really any different from the former.

Finally two other compound kinship correlations may be presented. They are of the type $r_{(a+b+c+d)\ (e+f)}$, that is the correlation between the summed ages at death of all the grandparents and the summed ages at death of both parents. The values of this coefficient are: for the Long. Series + 0.087 ± .035, and for the FHR Series + 0.060 ± .056. Neither significantly departs from zero correlation.

Summing up the whole case regarding parent-offspring correlations derived from the present data, we are led to essentially the same con-

TABLE 29

Theoretically Sensible Mid-parental-offspring Correlations in Respect of Longevity

CORRELATION IN RESPECT OF AGE AT DEATH BETWEEN	LONG. SERIES	NO. OF PAIRS
(Father's father + father's mother) and father. $r_{(a+b)e}$	+0.159±.034	365
(Mother's father + mother's mother) and mother. $r_{(c+d)f}$	+ .071±.035	365
Means	+.115	
	FHR SERIES	**NO. OF PAIRS**
(Father's father + father's mother) and father. $r_{(a+b)e}$	+0.058±.056	143
(Mother's father + mother's mother) and mother. $r_{(c+d)f}$	+ .105±.056	143
Means	+.082	

TABLE 30

Theoretically Nonsense Mid-parental-offspring Correlations in Respect of Longevity

CORRELATION IN RESPECT OF AGE AT DEATH BETWEEN	LONG. SERIES	NO. OF PAIRS
(Mother's father + mother's mother)[53] and father. $r_{(c+d)e}$	+0.037±.035	365
(Father's father + father's mother) and mother. $r_{(a+b)f}$	— .047±.035	365
	FHR SERIES	**NO. OF PAIRS**
(Mother's father + mother's mother)[53] and father. $r_{(c+d)e}$	+0.061±.056	143
(Father's father + father's mother) and mother. $r_{(a+b)f}$	— .126±.056	143

clusion as was reached by one of us [3] in an earlier study. The gist of this conclusion is that, for reasons unknown to us, the method of kinship correlation, regardless of the material to which it has so far been applied, leads to values of the correlation coefficients so low absolutely, and so variable amongst themselves both as to sign and as to magnitude, as to be out of accord with the now considerable and convincing body of other kinds of evidence, as well as elementary common observation, showing that heredity plays a really important rôle in the determination of individual longevity in man. It is our opinion that the biometric method of correlation, as it has hitherto been applied to the problem of the inheritance of longevity, is an inadequate and unreliable method so far as concerns this problem. Nothing in either the papers of Beeton and Pearson,[20,27,28], nor in the most recent work from the same laboratory [26] appears to furnish any ground for altering the opinion. We propose to continue our search to find out the reason for the discrepancy between the results given by the method of correlation and other methods, including the actuarial, in regard to the problem of inheritance of longevity. But at the same time we intend to get all the light we can on the problem by the use of other methods, which so far have yielded results which seem to us to be more trustworthy.

H. The Matings That Produced the Longevous

In common parlance, and upon the basis of the life table, the life duration of ancestors (that is of persons who survive to roughly at least 20 years of age) can fairly be thrown into three broad categories quantitatively, as follows:

A. Under 50 years.
B. From 50 to 69 years inclusive.
C. 70 years and over.

For convenience we shall designate persons falling in Class A as "Short-lived" in respect of age at death; those falling in Class B as "Average"; and those in class C as "Long-lived." Table 31 shows, in the two columns of figures on the left side the absolute and percentage frequencies of all the matings of parents which produced the propositi in the Long. Series—that is our 365 nonagenarians and centenarians—the matings being classified according to the above broad categories of age. The two right hand columns of figures give the same data for the matings producing the propositi of the FHR Series. The table is so arranged as to bring the percentage frequencies for the two series contiguous to each other.

TABLE 31

Matings Producing the Propositi in the Long. and FHR Series

NATURE OF MATING			LONG. SERIES MATINGS		FHR SERIES MATINGS	
Father		*Mother*	*Absolute frequency*	*Percentage frequency*	*Percentage frequency*	*Absolute frequency*
Short-lived	×	Short-lived	10	2.7	19.6	28
Short-lived	×	Average	8	4.9	18.2	3
Average	×	Short-lived	10			23
Short-lived	×	Long-lived	31	17.5	9.8	5
Long-lived	×	Short-lived	33			9
Average	×	Average	21	5.8	19.6	28
Average	×	Long-lived	51	23.3	21.0	15
Long-lived	×	Average	34			15
Long-lived	×	Long-lived	167	45.8	11.9	17
Totals			365	100.0	100.1	143

Table 31 shows clearly the striking difference in the character of the matings relative to longevity which produced the nonagenarians and centenarians on the one hand and the FHR propositi on the other hand. In the Long. Series there were proportionally nearly four times as many matings of the type Long-lived × Long-lived as in the FHR Series. Conversely there were over seven times as many matings of the type Short-lived × Short-lived involved in the production of the propositi in the FHR Series as in the production of the propositi in the Long. Series. Furthermore in the Long. Series there were but 5.8 per cent of the matings of the type Average × Average, as against 19.6 per cent of this type of mating in the FHR Series.

Figure 18 shows in graphic form the result of assembling the matings of Table 31 into three categories, according to whether they involved (a) no long-lived person, (b) one long-lived person, (c) two long-lived persons.

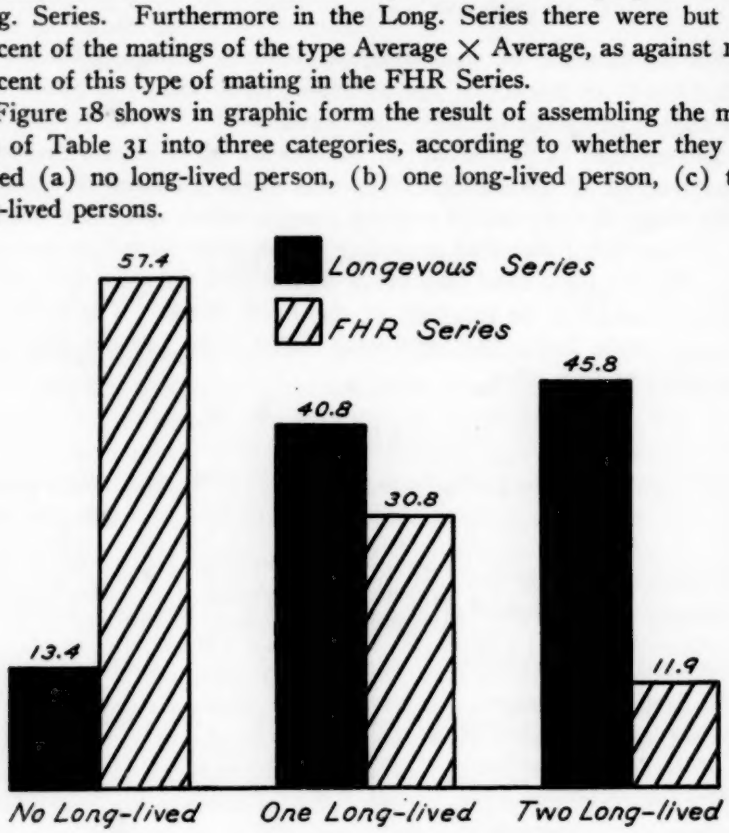

FIG. 18. BAR DIAGRAM SHOWING THE PERCENTAGES OF MATINGS OF PARENTS PRODUCING THE PROPOSITI WHICH INVOLVED (A) NO LONG-LIVED PARENT, (B) ONE LONG-LIVED PARENT, (C) TWO LONG-LIVED PARENTS IN THE LONGEVOUS SERIES (SOLID BARS) AND THE FHR SERIES (CROSS-HATCHED BARS)

It is evident that in the matings which produced the nonagenarians and centenarians the percentage of long-lived parentage *increases* as we pass from left to right in Figure 18, while conversely in the matings which produced the propositi in the FHR Series the percentage of long-lived parentage steadily *decreases* from left to right. Nearly 87 per cent of the parental matings producing nonagenarians and centenarians involved one or more long-lived persons, while in the FHR Series there were only about 43 per cent of such matings.

Let us next examine in the same way the matings in each series which produced the *parents* of the propositi. There will be two tables corresponding to Table 31, one for the matings producing the *fathers* of the propositi and one for those producing the *mothers*. Relative to the propositi these are *grandparental* matings that we shall now study. Table 32 gives the data for the matings producing the *fathers* of the propositi.

Table 32 exhibits a number of points of interest in comparison with Table 31. In the first place the percentage columns for the two series while still significantly different from each other are in some respects less different than in Table 31. That is to say, these grandparental matings are somewhat more nearly alike, for the two series, in respect

TABLE 32

Matings Producing the Fathers of the Propositi in the Long. and FHR Series

NATURE OF MATING			LONG. SERIES MATINGS		FHR SERIES MATINGS	
Father's father		*Father's mother*	*Absolute frequency*	*Percentage frequency*	*Percentage frequency*	*Absolute frequency*
Short-lived	×	Short-lived	5	1.4	0.7	1
Short-lived	×	Average	5	3.6	16.8	11
Average	×	Short-lived	8			13
Short-lived	×	Long-lived	15	7.7	11.9	9
Long-lived	×	Short-lived	13			8
Average	×	Average	20	5.5	6.3	9
Average	×	Long-lived	53	25.2	27.3	26
Long-lived	×	Average	39			13
Long-lived	×	Long-lived	207	56.7	37.1	53
Totals			365	100.1	100.1	143

of the longevities involved, than are the parental matings. This result is in accord with the earlier findings (Table 16, and Figures 13-15 *supra*) that the grandparents in the two series tended to be less differentiated from each other and from the general population than parents.

In both the Long. and FHR Series the percentages of matings of the Long-lived × Long-lived type are higher in Table 32 than in Table 31, by substantial amounts; at the same time the Short-lived × Short-lived matings are proportionately fewer in Table 32, particularly in the FHR Series, where there is only one (0.7 per cent) such grandparental mating.

Table 33 gives the data regarding the matings which produced the *mothers* of the propositi.

TABLE 33

Matings Producing the Mothers of the Propositi in the Long. and FHR Series

NATURE OF MATING			LONG. SERIES MATINGS		FHR SERIES MATINGS	
Mother's father		*Mother's mother*	*Absolute frequency*	*Percentage frequency*	*Percentage frequency*	*Absolute frequency*
Short-lived	×	Short-lived	3	0.8	3.5	5
Short-lived	×	Average	7	3.0	13.3	6
Average	×	Short-lived	4			13
Short-lived	×	Long-lived	21	12.1	18.2	15
Long-lived	×	Short-lived	23			11
Average	×	Average	16	4.4	9.8	14
Average	×	Long-lived	51	26.3	25.2	18
Long-lived	×	Average	45			18
Long-lived	×	Long-lived	195	53.4	30.1	43
Totals			365	100.0	100.1	143

Table 33 is plainly rather closely similar to Table 32. The breeding of the mothers of the propositi, in other words, is of about the same sort as that of the fathers so far as concerns longevity.

The significant differences between the breeding of the propositi, on the one hand, and their parents on the other hand, are clearly set forth for both Long. and FHR Series in Table 34, which summarizes Tables 31-33 inclusive.

TABLE 34

Summary of Tables 31-33 Inclusive

MATINGS PRODUCING	LONGEVOUS SERIES			FHR SERIES		
	Percentage of matings with			*Percentage of matings with*		
	No long-lived parent	*Only one long-lived parent*	*Two long-lived parents*	*No long-lived parent*	*Only one long-lived parent*	*Two long-lived parents*
Fathers of propositi	10.5	32.9	56.7	23.8	39.2	37.1
Propositi	**13.4**	**40.8**	**45.8**	**57.4**	**30.8**	**11.9**
Mothers of propositi	8.2	38.4	53.4	26.6	43.4	30.1

From the data presented we see:

1. That for two generations back of the propositi in the Long. Series (nonagenarians and centenarians) roughly something like one-half of all the individual matings have been composed of two Long-lived persons, the proportions being approximately 46 to 57 per cent of all matings concerned. In the breeding of the propositi of the FHR Series, on the other hand, there have been for two generations back only from about 12 to 37 per cent of all the matings concerned which have been composed of two Long-lived persons.

2. That only about a tenth (8.2 to 13.4 per cent) of all the individual matings for two generations back in the production of the nonagenarians and centenarians have involved no Long-lived persons. But in the FHR Series from 23.8 to 57.4 per cent of all the individual matings for two generations back of the propositi have included no Long-lived person.

3. The proportions of all individual matings involving only one Long-lived parent are in general roughly of the same order of magnitude in both the Long. and the FHR Series. But there are curious and orderly differences in detail. In the parental matings to produce the propositi the percentage of only-one-parent-long-lived matings is *higher* in the Long. Series (40.8 per cent) than in the FHR Series (30.8 per cent). But the opposite is true in the grandparental matings to produce the parents of the propositi, where the FHR Series has, in both cases, higher percentages of one-parent-long-lived matings than does the Long. Series.

IV. SUMMARY AND DISCUSSION

In this study we have analyzed and compared the ancestry in respect of their longevity of two groups of persons. One of these groups (called the Longevous or Long. Series) consists wholly of persons still living at ages of 90 years and above—83.8 per cent nonagenarians and 16.2 per cent centenarians in fact. The other group (called the FHR Series) consists of the oldest living siblings (except in a few cases where there were no living sibs and the oldest dead ones were taken) from sibships taken at random so far as concerns longevity. Both groups are alike in the fact that the six immediate ancestors (two parents and four grandparents) were dead and of known and recorded ages at death. In the present analysis no attempt has been made to differentiate between different causes of death of the ancestors, and in

particular there has been no effort to separate so-called "accidental" deaths from those generally regarded as non-accidental. Doubtless this will be regarded by some as a serious defect of the study. But whether it is so regarded or not will depend somewhat upon the point of view and upon an adequate apprehension of both the problem and the inherent difficulties in the case.[61] In this first study of the material our

[61] We have studied and thought about the causes of death for a good many years, and find it increasingly difficult to discover or define any sharp discontinuity between accidental and non-accidental causes. Consider, for example, Figure 9 *supra*. Is death from smallpox an accident or not? Is death in childbirth an accident? Because in this pedigree deaths from these causes occurred at young ages the tendency is to think of them as accidental, in the sense that they may have cut off the individuals concerned before they had had a chance to realize their innate biological potentialities of longevity. But does not the fact that some persons survive childbirth and some survive attacks of smallpox indicate that the constitution of the individual plays at least some part in the death or survival of those subjected to these particular biological stresses? Again consider pneumonia as a cause of death. A distinguished medical friend and correspondent always insists in discussions of this matter that all pneumonia deaths should be regarded as just as truly accidents as is being killed by an automobile. But on the other hand we have presented a considerable body of evidence [5(i)] indicating that the biological constitution of the individual plays a not unimportant rôle in determining whether he acquires and/or dies of pneumonia. Examples of the inherent difficulty, not to say impossibility of sharply defining and differentiating accidental deaths could be multiplied endlessly. Consider the case of the man who takes a trip and becomes infested with *Endamoeba histolytica* and subsequently dies from eating food prepared by a hotel cook who is a carrier. Such a death may from one point of view be regarded as accidental. But shall we, because such cases occur, regard intestinal amoebiasis generally as an accidental cause of death? Surely the weight of opinion would be against such an allocation. And so on through the whole list of infectious diseases. The same argument could be advanced for all of them. Furthermore it has been cogently argued by many that even deaths from being hit by an automobile are in some degree biologically selective. The argument is that it is the less alert, the less biologically fit, that are eliminated in this way. Obviously it is possible to push this type of argument too far, but it is difficult to deny that it contains some element of truth.

We have come to the conclusion that for the present at least it will be best to keep the study of the inheritance of human longevity on as completely an objective basis as possible, ruthlessly taking all accurately recorded ages at death at their face value, without any attempt to explain away by an appeal to accident any of them that may for theoretical reasons be uncomfortable. This position can furthermore be maintained with perhaps enhanced equanimity because it has been shown (cf. for example Stoessiger [36]) that when the so-called "accidental" deaths are alternatively included and excluded in the statistical analysis relating to inheritance of longevity there results no significant difference in any important conclusions.

judgment has been to present the data as they stand. If, as is the fact, they indicate when so presented *in toto* a definite and considerable influence of heredity in the determination of individual longevity the result is strengthened rather than weakened by the fact that accidental deaths among the ancestors are included.

What then are the more important and essential[62] results of this analysis? To us they seem to be those listed below. For the more detailed results the body of the text must be consulted.

1. The range of variation in TIAL (total immediate ancestral longevity, the summed ages at death of the six immediate ancestors of the propositi) is from 254 to 599 years. Both of these extreme figures occur in the Long. Series. In other words the whole range of variation in the TIALs of the FHR Series is included within that of the Long. Series (Table 9).

2. The mean and median TIALs of the nonagenarians and centenarians are over 60 years or nearly 16 per cent *greater* than those of the FHR Series, indicating statistically the importance of ancestral longevity in determining that of the individual (Table 10), even after all due allowance is made for the statistical consequences of the manner of selection of the FHR Series.

3. Not only is the mean TIAL greater in the Long. than in the FHR Series, but also the average age at death of every single ancestor (fathers, mothers, paternal and maternal grandfathers and grandmothers) of the group of nonagenarians and centenarians is greater than that of the corresponding ancestor of the FHR group (Table 15). The magnitudes of these average ancestral superiorities in longevity are of the order of 20 to 30 per cent in the case of the parents, and 10 to 14 per cent in the case of the grandparents (Figure 13). They are from 6 to 15 times their probable errors (Table 16) and therefore cannot be regarded as mere fluctuations of sampling.

4. It follows from paragraph 3 that since all of the individual ancestors taken separately are on the average definitely more longevous in the Long. than in the FHR group, any combination of them (as for

[62] We are fully aware of the fact that the present study does not analyze or discuss all the aspects of the material used. It is to be regarded solely as one stage or step in the development of our program of research on longevity. We expect to return later to the discussion of various problems upon which the present material is capable of shedding light, but which are omitted here from considerations of space.

example all male ancestors, all ancestors on the father's side, etc.) should show differences in the same sense. They do in fact (Tables 11, 13, 14).

5. The average longevity of the ancestors of the group of nonagenarians and centenarians appears to be generally greater than that of corresponding ancestors of persons not similarly selected for their own longevity (Table 17), and generally greater than that of comparable persons belonging to various general and special populations (Table 18).

6. But even though the statistical superiority of the ancestors of the group of nonagenarians and centenarians is clear and considerable, as has been pointed out in the preceding paragraphs, it is also an important fact that 13.4 per cent of the nonagenarian and centenarian group were bred from matings in which *neither* parent was Long-lived ("Long-lived" being defined as living 70 or more years). That is to say, that while 86.6 per cent of our group of highly longevous persons had either one or both parents Long-lived, there was a residue of about 1/7 of them who had no Long-lived parents. The same was true of 10.5 per cent of their fathers and 8.2 per cent of their mothers (Table 34). So it appears that extremely longevous persons *may* be bred from Short-lived or Average ancestry, in a small but not negligible proportion of all cases.

7. On the other hand 45.8 per cent of the nonagenarians and centenarians, 56.7 per cent of their fathers, and 53.4 per cent of their mothers had *both* of their parents in the Long-lived category (Table 34). These figures contrast with 11.9, 37.1 and 30.1 per cent for the FHR Series. Furthermore it must be noted that in the FHR Series 57.4 per cent of the propositi, 23.8 per cent of their fathers, and 26.6 per cent of their mothers had *neither* parent Long-lived. Altogether it appears that not only is the highly longevous group statistically superior in *average* ancestral longevity, but also they are bred from individual matings of marked superiority in respect of longevity.

8. The sibships to which the nonagenarians and centenarians belong are somewhat larger on the average than those to which the propositi in the FHR Series belong, and had experienced a definitely lower mortality up to the time of record than either the FHR sibships or the general population. The infant mortality (deaths under one year of age) in Long. Series sibships was only 5.86 per cent, as against 12.45 per cent in the life table population of 1901, and 14.35 per cent in the FHR Series sibships.

9. The mean total longevity (realized and expected) for the 2183 members of the sibships to which the nonagenarians and centenarians belong is 63.88 years per person, which is 14.64 years more than the expected mean duration of life at birth according to the standard life table used for comparison. The corresponding figures for the 906 members of the FHR Series sibships is 47.62 years, or 1.62 years less than the life table expectation of life at birth, an insignificant difference.

10. Comparing the two groups here dealt with it appears that each additional 3.7 years in mean TIAL per pedigree has associated with it one additional year in mean duration of life of the offspring, and this without any allowance or correction whatever for accidental deaths.

11. All possible homogamic longevity correlations in the ancestry of both groups were investigated. The coefficients were generally too low to be of any practical significance, and furthermore, varied widely among themselves.

12. The same was true of the kinship (ancestral) longevity correlations within each group.

13. In the group of nonagenarians and centenarians, 41.4 per cent are moderate drinkers. This is 4.1 per cent *more* than was found in a random sample of the general population of average longevity. Furthermore 3.7 per cent of the nonagenarian and centenarian males are heavy drinkers. While this percentage is less than that of heavy drinkers in the sample of the general population with which they were compared, it is significant that there are as many as 3.7 per cent of these extremely longevous males who have been heavy drinkers. The evidence from this study clearly indicates that the moderate use of alcoholic beverages throughout life is not incompatible with the attainment of great longevity.

14. More than half (56.6 per cent) of the nonagenarian and centenarian group were total abstainers, as compared with 45.3 per cent in the sample of the general population used for comparison.

15. There is no evidence of discontinuous variation in the distribution of TIAL, or in the distribution of ages at death of any one of the six immediate ancestors of the propositi in either group.

Taking all the evidence presented in this study as a whole it would seem to leave no doubt as to the importance of heredity in the determination of the longevity of the individual human being. Comparison of the two groups of persons here dealt with shows that for every year

of superior longevity realized by the longevous group (and their sibs) there is a corresponding superiority in the longevity of their immediate ancestors, not so large in amount to be sure because the principle of regression operates, but still real and larger than can be accounted for by chance fluctuations of sampling. Furthermore this result appears in spite of the fact that no allowance or correction has been made at any point for accidental deaths. Failure to make any such allowance or correction plainly works *against* the expression of any hereditary factor in longevity. In short the hereditary influence in longevity shown in this study is stated quantitatively as a minimum for the actual families concerned.

The data presented in this study do not, in our opinion, permit the formation of any critical judgment as to the mechanism of the inheritance of longevity, or even as to whether it is Mendelian in nature or otherwise. We are of the opinion that really critical evidence on this point cannot be derived from the purely mathematical procedure of fitting complex Mendelian formulas to statistical data of the sort presented here, or elsewhere available in such studies as have hitherto been made on the problem. After such a mathematical procedure has been successfully carried out it proves nothing more than that it has been carried out successfully. It demonstrates nothing whatever as to the *reality* of the mechanism postulated. It merely shows that the observed results *may have been due* to the postulated mechanism. But showing that a phenomenon *may have been* caused by X is a wholly different matter than showing that it *was* caused by X.

We are by no means of the opinion that it is inherently or necessarily impossible to discover and critically prove the existence of a Mendelian mode of inheritance of longevity in man, if it really does exist. On the contrary our program of investigation is definitely planned towards such an end. But it will be necessary to resort to a different kind of evidence than that presented in this, or any other similar statistical study. We believe that we have in the archives of our laboratory material that will furnish the necessary kind of evidence, and in due time we hope to be able to analyze it from this point of view. But in the meantime we are endeavoring to maintain a completely open and sceptical mind as to the ultimate outcome. We find ourselves, in short, in complete agreement with Russell [63] in the opinion that there

[63] Russell, E. S. The Interpretation of Development and Heredity. A Study in Biological Method. *Oxford* (Clarendon Press) 1930. Pp. [4 leaves] + 312.

is as yet no compelling reason to suppose that *all* phenomena of heredity in the organic world necessarily follow the Mendelian plan. From one point of view Russell states the case compactly in the following passage (pp. 68-9):

> The extension of the Mendelian principle to characters other than those which can be studied in actual crosses is of course based on the general conception that the chromosomes constitute the physical basis of heredity. The argument runs somewhat as follows. The Mendelian factors are certainly borne by the chromosomes, and their behaviour can be interpreted by what we know of the distribution of the chromosomes in maturation, reduction, and fertilization. The chromosomes appear to be the only possible vehicle for the transmission of the characters of the organism as a whole, since they are the only equivalent structures in the male and female gametes, and the contribution of both parents to the hereditary equipment of the offspring appears to be equal. Hence it would seem that what is true of one set of characters should be true of the others, since all are borne by the chromosomes. We arrive therefore at the conception of a genetic constitution, consisting of a large number of factors, like those demonstrated by Mendelian research, and responsible between them—with the coöperation of the cytoplasm and of environmental conditions—for the inheritance and development of all the characters of the organism.
>
> There is, however, contained in this argument a curious *petitio principii*, which has as a rule escaped notice, though recently it has been pointed out by Winkler. The logical slip is the assumption that the paternal and maternal contributions are of equal importance. For how can this be proved? In all breeding experiments we can deal only with differences, and only with such differences as are not incompatible with fertile inter-crossing. The great bulk of characters is necessarily common to both parents, and about each parent's contribution to the inheritance of what is common to both, breeding experiments can obviously tell us nothing. Experimental work can deal only with the inheritance of minor characters or of minor variations of major characters. Accordingly, so far as the facts go and strict logic carries us, inheritance of the main characters might be purely maternal, and involve not only the nuclear apparatus but very intimately the cytoplasm, which is practically absent in the majority of male gametes. The facts under consideration do not of course prove this unilateral inheritance, but they can certainly not be used to demonstrate that the main characters are derived in approximately equal measure from both parents. This being so, the logical argument for the extension of the gene theory to *all* characters of the organism falls to the ground, and the question remains completely open.
>
> Here then is the point of Johannsen's suggestion, that in addition to all the separable, mendelizing characters, which can be treated in terms of separate factors, there must be postulated a "great central 'something,' as yet not divisible into separate factors." That it will ever be so divisible seems highly improbable.

We must conclude then, until further evidence is forthcoming—if it ever is—that the scope both of the Mendelian principle and of the gene theory is limited to such rather superficial variations as can be dealt with by experimental breeding. The direct contribution which genetic experiment and genetic theory make to the main problem of heredity—the reproduction of specific type, apart from minor deviations—is therefore a strictly limited one; the fundamental problem is in fact hardly touched.

Many years ago, when the modern gene theory was still in its earliest infancy, one of us made essentially the same point [64] as is now made by Russell. The passage will bear quotation here (pp. 7-8):

. . . . . two definite inferences are not only plain, but indeed inescapable. They are:

1. That germinal substance is innately possessed of a definite and particular *specificity*, which reaches, in degree, to the order of the individual, and which finds its most obvious objective expression in the specificity of somatogenesis; and

2. That the processes of reproduction are of such a sort as to tend to maintain this specificity from generation to generation.

In the light of this summary analysis of elementary facts it seems clear that *the critical problem of inheritance is the problem of the cause; the material basis; and the maintenance of the somatogenic specificity of germinal substance.*

. . . . . there are clearly to be distinguished different orders or degrees of the somatogenic specificity of the germinal substance. This fact has led to a good deal of confusion in the use of the term "heredity." For example, it is no doubt entirely correct to say that birds have feathers because this sort of dermal covering is hereditary in the class of animals specified. But heredity in this sense means, both theoretically and practically, a very different thing than when the equally true statement is made that a Barred Plymouth Rock female inherits the barred color pattern of her feathers from her sire only, and not at all from her dam. In the first case one is dealing with a phyletic matter, in the other case with a subvarietal. It is obvious that the degrees of germinal specificity which determine the two sorts of hereditary phenomena indicated in the example must be of widely different orders. One represents the substratal or general element of heredity, the other the more superficial or individualistic element. It is obvious that the number of distinguishable different orders of germinal specificity is as great as the number of distinguishably different orders of variability, which in turn is more or less closely reflected in the various subdivisions or stages in taxonomic classification.

Longevity (duration of life) would appear to be biologically a rather fundamental attribute of the organism. Indeed it may reasonably be regarded as a single numerical expression of the integrated effects of all the forces that operate upon the individual, innate and environmental.

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[64] Pearl, R. Modes of Research in Genetics. *New York* (Macmillan) 1915. Pp. viii + 182.

It not only may be, but is in fact, affected adversely or favorably by environmental circumstances of the most varied sorts,[65] but also it is closely bound up with the biological *constitution* of the individual, and biological constitution in man is a very highly complex matter indeed.[66] These considerations, coupled with the absence of evidence for any discontinuities of variation in its expression however analyzed hitherto, lead us to maintain for the present an open mind as to whether longevity in man is really inherited according to any Mendelian theory, however complicated. Finally it is well always to remember that Mendelian inheritance is *au fond* a theory of *alternative* inheritance.

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[65] For a variety of experimental and statistical evidence on this point see the following two books by R. Pearl: The Rate of Living. *New York* (Alfred Knopf) 1928. Pp. [8 leaves] + 185; Studies in Human Biology. *Baltimore* (Williams and Wilkins Co.) 1924. Pp. 653; and other references therein cited.

[66] Pearl, R. Constitution and Health. *London* (Kegan Paul) 1933. Pp. 97.

ORGANIZATION FOR RESEARCH IN POPULATION

I

THE FOUNDATION, HISTORY AND PURPOSE OF THE INTERNATIONAL UNION FOR THE SCIENTIFIC INVESTIGATION OF POPULATION PROBLEMS

IN the fall of 1927 there was held at Geneva a world population congress at which twenty-seven countries were represented. At this congress a committee was appointed to consider the formation of a permanent organization for the purpose of securing international scientific coöperation in the study of population problems. As a result of the deliberations of this committee a general assembly of delegates of the proposed permanent organization met in Paris in 1928 and passed upon a body of statutes of the proposed union. Dr. Raymond Pearl was elected first President and it was decided to hold the Second General Assembly in 1931. This Second General Assembly met in London in June of the year appointed, amended and revised the statutes and held an International Conference at which representatives of all the adhering National Committees, with the exception of Italy, were present. Sir Charles Close was elected President in succession to Dr. Raymond Pearl.

The Objects and Work of the Union

The objects of the Union are to organize and encourage research, whether depending upon international coöperation or within the separate national units, to co-ordinate such researches, and to hold a General Assembly, and, if desired, an International Congress, every three years. The Union is bound to "confine itself strictly to scientific investigations and to refuse to enter upon religious, moral or political discussions, or as a Union, to support a policy regarding population of any sort whatever, particularly in the direction either of increased or diminished population."

At a time such as the present it surely is unnecessary to point out the importance of population studies carried on from a wholly unbiased

objective scientific point of view. We have in certain parts of the world a rapidly growing population, densely distributed over restricted areas, which raises a very definite practical problem regarding material provision for the future of its members. In other parts of the world a rapidly declining birthrate seems to point to problems of an opposite character facing us in the near future. Over and above all this is the world wide economic depression from which we are perhaps on the point of emerging, but which suggests that at best we must be prepared for repetitions of similar experiences in the near future unless entirely new ways of managing international affairs shall be devised. The problems here involved are distinctly also population problems, if we understand this term broadly to include problems arising from the relation of human populations to their environment.

Obviously the population problem as a whole has many ramifications. Three of its aspects have been made the subject for study of three special commissions, namely, Commission I on Population and Food Supply, Chairman, Professor J. D. Black of Harvard; Commission II, Fertility, Fecundity and Sterility, F. A. E. Crew of Edinburgh; Commission III is temporarily in abeyance; its topic was The Vital Statistics of Primitive Races. Into its place has stepped a fourth Commission for which the name "Commission on Ethnogenics" has been suggested. It is to deal with ethnogenic units in relation to milieu including cultural factors. The plans of work for this Commission are not yet mature.

The Union operates essentially through a collection of National Committees formed in each of the countries represented in the Union, and each committee promoting and coördinating, in the country which it represents, the investigation of scientific questions relating to population, more especially in relation to international needs. Among the means through which these National Committees are empowered to operate is the awarding of Fellowships and Grants in Aid of Research allotted from such funds as they may have at their disposal through the power conferred on them by the Union to receive and administer funds for these purposes.

The American National Committee took its origin early in 1931 upon the invitation from Dr. Raymond Pearl, at that time President of the Union, requesting Dr. Louis I. Dublin to take the necessary steps for its formation. It held its first meeting on February 4, 1931 and has since been active in promoting research on population problems in the

United States. It was well represented at the Second General Assembly of the Union, which met in London in June, 1931, and to which members of the committee contributed eight papers, the result of recent work on various phases of the population problem. Through the liberality of the Rosenwald Fund the committee was able to grant two fellowships and to give minor financial aid to a third investigation. The committee took active part in the formation of the Population Association of America, and by mutual agreement was designated as the Committee on International Relations of that organization.

The work of the Union, in addition to that conducted by the special commissions and under the auspices of the National Committees, includes the organization of International Congresses in connection with its General Assemblies. Such a congress would undoubtedly be planned for the Third General Assembly which was scheduled to meet in 1934. The plans for this have not yet been developed.

II

POPULATION ASSOCIATION OF AMERICA

In the Fall of 1930 a number of population workers in the United States felt that the time was ripe for the formation of a national organization which should promote research in population in this country, and should maintain some sort of coöperation with the International Union for the Scientific Investigation of Population Problems. To this end a preliminary conference was held in New York on December 15, 1930. It was attended by thirteen persons, and the whole question was discussed thoroughly. There was unanimous agreement that such an organization should be formed, and by vote a committee was authorized, and later appointed, to proceed with the steps necessary for organization. At this time the American Committee of the International Union was being organized, and it was voted that if a population association were eventually formed some basis of affiliation between the two should be worked out.

Pursuant to the foregoing action a Second Conference was called on May 7, 1931. It was attended by thirty-eight representative students of population questions. After extended deliberation and discussion it was voted to organize such a body, to be called the Population Association of America, the purpose of which, as eventually included in the Constitution, was to be "research in respect to problems connected with

human population, in both its quantitative and qualitative aspects, and dissemination of the results of such research." Officers for the first year of provisional operation were elected as follows:

President—Henry Pratt Fairchild
Vice-President—William F. Ogburn
Second Vice-President—Robert R. Kuczynski
Secretary-Treasurer—Alfred J. Lotka
Executive Committee:
O. E. Baker, Niles Carpenter, Louis I. Dublin, Ellsworth Huntington, Clarence C. Little, Frederick Osborn, Raymond Pearl, Leon E. Truesdell, A. B. Wolfe.

During the ensuing year a Constitution * was drafted which was presented to the first regular Annual Meeting of the Population Association on April 22, 1932. After full discussion it was adopted including, as Article IV of the By-Laws, a statement worked out by a joint committee of the Population Association and the American Committee of the International Union as follows: "The American National Committee of the International Union for the Scientific Investigation of Population Problems is designated as the Committee on International Relations of the Population Association of America, it being understood that the American National Committee retains its autonomous character."

This Constitution provides for two types of members, one a general membership open to any interested person, the other a College of Fellows composed of students of recognized scientific standing. The general membership is represented on the Board of Directors, which is so organized, however, that the actual direction of the affairs of the Association lies ultimately in the College of Fellows.

Public sessions, presenting papers and discussions, were held in conjunction with the Eastern Sociological Conference. Officers were elected as follows:

President—Henry Pratt Fairchild
First Vice-President—William F. Ogburn
Second Vice-President—Warren S. Thompson
Secretary-Treasurer—Clarence G. Dittmer

* Copies of this Constitution may be secured by writing to Population Association of America, Washington Square East, New York City.

Board of Directors:

O. E. Baker, J. D. Black, Edmund de S. Brunner, Niles Carpenter, Robert E. Chaddock, Louis I. Dublin, L. B. Dunham, Frank H. Hankins, Ellsworth Huntington, Frederick Osborn, Raymond Pearl, Harold A. Phelps, Lowell J. Reed, Leon E. Truesdell, A. B. Wolfe.

A committee was appointed, instructed to confer with a similar committee of the American Committee of the International Union, to consider a practical working basis of relations between the two bodies. This committee met, and decided that an essential step was the formulation of an outline of the field of population research. The Outline presented in this document is the result of this decision.

The Second Annual meeting of the Association was held on May 12, 1933. The existing officers and Board of Directors were re-elected. The public program on this occasion was offered in conjunction with the American Statistical Association.

III

OUTLINE OF THE FIELD OF RESEARCH IN POPULATION

The following Outline, prepared under the auspices of a Joint Committee of the Population Association of America and the American Committee of the International Union for the Scientific Investigation of Population Problems, is made public in the hope that it may prove of value and assistance to students of Population in all of its branches. It is intended to be merely tentative and suggestive. Many of its sections are capable of expansion into almost unlimited detail. In the Outline as presented, the subdivisions are carried much further in certain sections than in others. This is due partly to the greater familiarity of those who prepared the Outline with some departments than others, and partly to the hope that the more detailed classifications would encourage similar analysis in other departments by those competent to undertake it. Suggestions for the improvement and amplification of the Outline will be gratefully received by the officers of the Association.

The Outline concerns itself exclusively with research. Even in the applied branches, the subject matter is research into purposes and methods of application. This Outline is not designed as a guide to social workers, and has nothing to do directly with the actual, active achievement of results.

A population, as understood in this Outline, is a group of men, women, and children, coördinated into that type of community ordinarily designated as a "society." The concept of population has a range corresponding to that of the concept of society. It may run from relatively small units at one extreme to the total of humanity at the other extreme.

The main object of this Outline, as of the two organizations that sponsor it, is the promotion of scientific research in the field of population. To this end, it is designed to assist individual workers to understand the nature and scope of the field of population, to organize their own studies, to correlate them with the work of other students, and to visualize needed research in terms of definite projects. The Population Association at the present time has no funds whatever to assist or subsidize research projects. Nevertheless, it realizes that the outlining of projects at the present time is of great importance. Consequently, it will welcome information as to projects already under way or contemplated, and others which in the minds of the correspondents could profitably be undertaken in any of the fields indicated in the Outline. All communications should be addressed to Population Association of America, Washington Square East, New York City.

OUTLINE OF THE FIELD OF RESEARCH IN POPULATION

Section I. Collection and Presentation of Facts. Demography.

I. Collection
  A. Larithmics. (See Note 1). Quantitative Data.
    1. Static. Size of Population.
      a. Enumeration
        (A) Censuses
          Local
          National
          International
        (B) Gross numbers
          Distributed by
            Age
            Sex
            Race
            Color
            Nationality
            Nativity

Occupation
Wealth
Income
Education
Marital status
Religion
Political affiliation
Etc., etc.

2. Dynamics. Growth of population.
   a. By natural increase
      (A) Vital statistics
         Birth rates
            Classified as in 1. a. (B)
         Death rates
            Classified as in 1. a. (B)
         Rates of population growth. (See Note 2)
            Classified as in 1. a. (B)
   b. By migration. (Classified as in Sec. II, III)
      Increments. Immigration
      Decrements. Emigration

B. Eugenics (See Note 3) Qualitative Data. (Classified as in Sec. II, II)
   1. Somatic traits
   2. Intelligence
   3. Personality
   4. Moral traits
   5. Social traits

II. Presentation and Illustration
   A. Descriptive
   B. Pictorial
      Still
      Moving
   C. Statistical
      Graphs
      Tables
      Charts
      Indexes
      Curves
      Formulas
      Etc., etc.

Section II. Analysis and interpretation of facts
Population theory: laws and principles.

I. Larithmics. Factors in the numerical growth of population.
   A. Extrinsic
      1. Geographic factors. The earth. Land

a. Area. Standing room
   (A) Water
   (B) Dry land
      Topography
b. Productivity. Natural resources for food and material supplies
   (A) Water
      Salt
      Fresh
   (B) Dry land
      Topography
      Climate
         Temperature
         Precipitation
         Winds
         Sunshine
      Soil qualities
         Depth
         Age
         Composition
         Texture
      Natural supplies
         Minerals
         Flora
         Fauna

2. Economic factors. Economic culture.
   Arts of life. Artificial developments and improvements of 1.
   a. Economic
      (A) Arts of life
         (1) Stages of culture
            Hunting
            Animal breeding
            Agriculture. Mining
            Mechanical industry
         (2) Intellectual equipment
            Dexterities
            Trade technique
            Applied science
         (3) Capital accumulations
            Tools, machines, implements, plants
      (B) Standard of living
         (1) Negative. As an alternative to numerical growth
         (2) Positive. As an aid to numerical growth

3. Sociological factors
   a. Institutional factors
      (A) Marriage systems
         Polygamy
            Polygyny

Polyandry
Monogamy
(B) Age of marriage
Trends and causes
(C) Marriage rate
Trends and causes
(D) Divorce and divorce rates
Trends and causes
(E) Illegitimacy
b. Cultural factors
(A) Education
Of men
Of women
Segregation and coeducation
(B) Occupation
Affecting fecundity and fertility (See Note 4)
Of men
Of women
(C) Home
Type of domicile
Rooms—number, arrangement, uses, etc.
Sleeping arrangements
Breadwinners
(D) Recreation
In the home
Out of the home
Theaters
Movies
Dance halls
Recreation centers
Parks
Automobiles
Road-houses
Etc., etc.
(E) Moral code
Double standard
Chastity
Modesty
Chaperonage
Petting
(F) Type of community
Rural
Small town
City
c. Artificial control
(A) Social—enforced

Segregation
Sterilization

(B) Personal—volitional, but made possible by social developments, and tolerated, permitted, or sanctioned by society.
Birth control

(1) Abstinence
Celibacy
Monastic or religious
Secular
Marital continence

(2) Abortion

(3) Contraception

(C) Institutional
Infanticide
Ceremonial killing of the old

B. Intrinsic

1. Biological factors

a. Natality. Births (See Note 4)

(A) Fecundity. Physiological capacity to participate in reproduction

(B) Sterility or infecundity. Lack of fecundity

(C) Fertility. Fecundity expressed in performance

(1) Potential
Egg-producing capacity of females
Sperm-producing capacity of males
Frequency-ability of females to conceive and bring offspring to birth

(2) Realized. Compared with potential, determined or presumptive. If less, causes

(a) Group natality. Birth rate.
Affected by factors listed in Sec. II, I A.

(b) Family natality. Births per couple. Limiting factors: primary—inherent in one or both of mates; secondary—incompatability of mates.
Physical, including still-births
Disease
Venereal
Other
Structural
Functional
Mental and emotional
Psychoses, fear, etc.
Traditional norms
Prudential considerations

b. Vitality. Capacity to survive. Viability
Health
Energy
Tenacity of life
Expectation of life

c. Mortality. Deaths. Almost universally regarded as undesirable. Therefore all social attention focussed on causes and prevention.
(A) Causes
(1) Disease
Types
Causes
Cures
Prevention
(2) Accident
Industrial
Recreational
Domestic
Etc., etc.
(3) Homicide
Murder
Suicide
War
(B) Prevention
Whole range of medical, hygienic, and public health activities. Belongs primarily in Section III, I.

2. Psychological, habitual factors. Influenced by conventions and traditions.
a. Psychological impulse
Sex urge
Nature and basis
Stimuli
b. Sex habits
(A) Pre-marital
(1) Chastity
(2) Indulgence
Heterosexual
Promiscuity
Various illicit relationships
Homosexual
Autoerotic
(B) Marital
(1) Frequency of intercourse
(2) Habits of intercourse

II. Eugenics and Euthenics. Factors in qualitative determination of population.
A. Extrinsic factors. Environment.
1. Geographic
Classified in Section II, I A 1

2. Economic
   Classified in Section II, I A 2
3. Sociological. Includes whole social structure.
   Partially classified in Section II, I A 3

B. Intrinsic factors. Heredity.
   1. Human genetics. Physical mechanism of human inheritance
      (A) Organic
      (B) Plasmic
         Theory of genes, chromosomes, unit characters, etc.
         Mendelianism

C. Combined results of these factors in human personality
   1. Analysis, classification, and cataloguing of human traits.
      (A) Somatic
         (1) Color
            Skin
            Hair
            Eyes
         (2) Form
            Body
            Hair
            Cranium
            Features
               Eyes
               Nose
               Mouth
               Etc., etc.
         (3) Proportions
         (4) Susceptibilities and immunities
         (5) Anomalies and aberrations of all kinds
      (B) Intellectual
      (C) Emotional
      (D) Moral
      (E) Social
      (F) Spiritual
   2. Assignment of these traits, absolutely or relatively, to origin in heredity, or environment in its various aspects.
      Methodology of testing and analyzing human personality. Studies of growth, and effect of different environments.
         Physical tests
         Intelligence tests
         Character tests
         Social adequacy tests
         Psychoanalysis
         Studies of twins
         Studies of foster children

III. Migration. Inter-group factors in population change, larithmic, euthenic, and eugenic.
  A. Intrasocietal
    1. Region to region
      a. Causes
      b. Processes
      c. Effects
    2. Rural to urban
      a. Causes
      b. Processes
      c. Effects
    3. Urban to rural
      a. Causes
      b. Processes
      c. Effects
  B. Intersocietal
    1. Emigration
      a. Causes
      b. Processes
      c. Effects
    2. Immigration
      a. Sources
      b. Composition
      c. Causes
      d. Processes
      e. Regulation
      f. Effects

IV. Mathematical Theory of Population. An interpretation of population growth and change, quantitative and qualitative, in terms of mathematical and statistical sequences. (To be expanded.)

Section III. Application. The use of scientifically determined theory, laws, and principles for the promotion of recognized social values and the attainment of accepted social objectives. Rules of social administration. Policies and programs. (See Note 5)

I. Practical or Applied Larithmics.
  A. The relation of size of population to human welfare
    1. Criteria
      a. Optimum population
        (A) Criteria
          (1) Economic. Standard of living
            Relation of population growth to standard of living
              Favorable
              Antagonistic

(2) Aesthetic
Art
Recreation
(3) Nationalistic
Militaristic
Prestige
(4) International
Relation to world peace
(5) Moral
Effect on vice, crime, etc.
(6) Religious
(B) Methods of measurement and forecast
b. Underpopulation
(A) Criteria as in a. (A)
Causes
Cures
c. Overpopulation
(A) Criteria as in a. (A)
Causes
Cures
2. Control
a. Stimulation
(A) Increasing births
Education and propaganda
Rewards and exemptions
(B) Decreasing deaths
Medical, sanitary, hygienic, and preventive measures of all kinds
b. Restriction
(A) Decreasing births
(1) Coercive
Sterilization
Segregation
Marriage requirements
Penalities for illegitimacy
(2) Persuasive
Education and propaganda
Public sentiment, ridicule, etc.
(3) Permissive
Social sanction of birth control
Provision of facilities for birth control
Discovery and invention of safe and effective contraceptives
Provision of access to contraceptive information, advice, and supplies. Physicians, clinics, etc.

(B) Increasing deaths. Last resort. Seldom deliberately applied internally by civilized societies
War

c. Migration
To increase population
To diminish population

II. Practical or applied Euthenics. This includes practically the whole field of social engineering and social reform and is too complex to be analyzed here.

III. Practical or applied Eugenics. The suppression, elimination, selection, and intensification of given traits, as far as they are due to heredity, according to recognized norms. The breeding of the ideal man.

A. The establishment of a breeding program in accordance with the acknowledged values and objectives of the society in question, such as social continuity, longevity, health, beauty, intelligence, happiness, social responsibility, industry, economic efficiency, etc., etc., in accordance with principles indicated in Section II, II C.

B. The development and application of methods to put this breeding program into effect. Negative and Positive Eugenics.

1. Securing conformity on the part of individuals

a. Dissemination of eugenic ideal
Education
Propaganda
Religion

b. Arrangement of matings on eugenic principles
Negative
Coercive
Marriage restrictions
Segregation
Persuasive
Positive
Coercive
Persuasive

c. Regulation of size of families
Negative
Permissive. Social sanction of birth control
Coercive
Sterilization
Persuasive
Positive
Permissive. Social sanction of birth control
Coercive. Almost impossible
Persuasive
Awards, subsidies, prizes, exemptions, etc.
Social recognition, acclaim, prestige, etc.

NOTES

Note 1.—For the origin, derivation, and meaning of the word "Larithmics," see Fairchild, Henry Pratt, Larithmics, *American Journal of Sociology*, 36:2:278-81, September, 1930.

Note 2.—The present incompleteness of the science of Larithmics is illustrated by the fact that there is no accepted technical term to indicate changes in the size of population resulting from the balance of births and deaths. Customarily, some phrase is used including the word "growth" or "increase." This is unsatisfactory, as the change may be downward instead of upward.

Note 3.—The words "Eugenics" and "Euthenics" are intrinsically ill-suited for scientific classification, because the prefix "eu," meaning "well," implies a value judgment, which has no place in the field of theoretic research, and even in the applied field should not be presupposed or taken for granted. Research in population should be just as much concerned with bad origins as good origins, and in fact should not pass judgment upon its material as to its goodness or badness. But the word "eugenics" is well established in scientific, as well as popular usage, and the word "euthenics" is harmonious with it, and accordingly it seems best not to attempt to alter this particular terminology at the present time.

Note 4.—The terms "Fecundity" and "Fertility" in this Outline are used in correspondence with definitions recommended by a committee, appointed for the purpose, to the Population Association of America at its Annual Meeting on May 12, 1933, and adopted by the Association for a year's provisional trial. These definitions, along with those of related terms, are as follows:

NATALITY—The birth performance of a group, correlative to mortality; natality rate is synonymous with birth rate.
FECUNDITY—Physiological capacity to participate in reproduction.
STERILITY (or INFECUNDITY)—The lack of physiological capacity to participate in reproduction.
FECUND—Having fecundity.
STERILE (or INFECUND)—Lacking fecundity.
FERTILITY—Fecundity expressed in performance and therefore measurable; virtually synonymous with natality.
INFERTILITY—Absence of fertility; synonymous with childlessness.
FERTILE—Expressing fertility.
INFERTILE—Manifesting infertility; synonymous with childless.

Note 5.—In considering the practical or applied section of this Outline, particular care should be exercised to remember that it is an outline of research. Although this section deals with the practical aspects of population theory, there is no intention to prescribe therein what a society *ought* to do about its population. It is designed to indicate the various avenues of research into the bearing of different social developments, and different types of social activity and programs, upon certain recognized features of social welfare or progress. But this Outline does not undertake to set up these objectives or criteria, or to pass critical judgment upon them, or to indicate how they are to be established or tested. As far as

goals, objectives, or value judgments are implied in this Outline they are assumed *prima facie*. They are broad desiderata that are generally accepted by common consent by the great majority of the members of society, at least western society; such as health, vigor, intelligence, economic capacity, trustworthiness, etc., etc.

The basic reason for this is that ultimate values can never be scientifically justified. Their existence may be proved, but not the rightness or legitimacy of their existence. That is, science may demonstrate that the mores of a given society are animated by certain basic objectives or values, but it cannot prove, or disprove, that these values are right or should prevail.

At the same time, it should be recognized that, once the ultimate values are given, assumed, or specified, the application of social theory to these values may be just as scientifically treated as the derivation of theory.* Furthermore, since any scientific and constructive social policy or program must be based upon some recognized and accepted final values, there is great need in the field of population for research into the values that are actually held by contemporary societies, and the relationship of these values to each other. Without positive knowledge on this point all statements of applied theory must be purely provisional and tentative. Yet, unfortunately, in the field of population, most of the objectives upon which practical policies are based are taken for granted, even by scientific workers. We make certain elementary assumptions about the desirability of health, longevity, and intelligence, and then proceed to discuss programs of larithmic or eugenic control as if we knew not only what would be best for society, but also what each society *believes* would be best for itself. As a result, much of the discussion ends, at best, in vagueness, and at worst in contradiction and confusion.

As a single example may be taken the question of a numerically increasing population. Probably the majority of people in the United States regard growth in population as intrinsically desirable. Probably even more value a high material standard of living. But who knows which of these desiderata is more highly prized, which takes precedence over the other? Consequently, suppose a scientific population worker should demonstrate conclusively that at the present time an increasing population is prejudicial to the standard of living, and vice versa, how could he know what program to recommend to his society? Before he can advise scientifically, he must know how these values are actually rated among his contemporaries.

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* A fuller statement of these principles may be found in Fairchild, Henry Pratt, The Possibility, Character and Functions of Applied Sociology, *Social Forces*, 11: 2: 182-187, December, 1932.

NOTES

A CENTRAL MALARIA LIBRARY

A MALARIA library was founded in Rome by the Stazione Sperimentale per la Lotta Antimalarica in 1925, and an "Index to Malaria Literature" is issued annually by the Station.

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A. MISSIROLI
(DIRECTOR)

RECENT LITERATURE

USEFUL IN THE STUDY OF HUMAN BIOLOGY

In this department will be noticed the books and reprints which are currently received at the Editorial Office. Publications intended for notice here should be addressed to Dr. Raymond Pearl, Department of Biology, School of Hygiene and Public Health, 1901 East Madison Street, Baltimore, Maryland, U. S. A.

I. EVOLUTION AND GENETICS

1. *Evolution—General Principles*

BRANSON, E. B., and M. G. MEHL. Conodont Studies Number 1. *The University of Missouri Studies*, Vol. VIII, No. 1. *Columbia* (University of Missouri), 1933. Pp. 1-72 + 4 plates. 7⅜ × 10 inches. $1.25 (paper).

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